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TRANSACTIONS

OF THE

AMERICAN CLIMATOLOGICAL ASSOCIATION.

FOR THE YEAR 1913.

VOLUME XXIX.

"The object of this Association shall be the study of Climatology and Hydrology and of Diseases of the Respiratory and Circulatory Organs."—*Constitution.*

PHILADELPHIA:

PRINTED FOR THE ASSOCIATION.

1913.

NOTICE.

The Association assumes no responsibility for the statements and opinions expressed in the papers read at its meetings.

OFFICERS OF THE ASSOCIATION.

Presidents.

CHARLES L. MINOR, M.D., Asheville, N.C., 1913.
JAMES M. ANDERS, M.D., LL.D., Philadelphia, 1914.

Vice-Presidents.

JAMES M. ANDERS, M.D., LL.D., Philadelphia, 1913.
CHARLES D. ALTON, M.D., Hartford, 1913.
LAWRASON BROWN, M.D., Saranac Lake, 1914.
WILL HOWARD SWAN, M.D., Colorado Springs, 1914.

Secretary and Treasurer.

GUY HINSDALE, M.D., Hot Springs, Va.

Representatives on the Executive Committee of the Congress.

*Dr. ROLAND G. CURTIN, Philadelphia.
Dr. THOMAS DARLINGTON, New York, *Alternate.*

Committee of Arrangements, 1914.

Dr. PHILIP MARVEL, Atlantic City.
Dr. W. E. DARNALL, Atlantic City.

Council.

CHARLES E. QUIMBY, M.D., New York.
EDWARD R. BALDWIN, M.D., Saranac Lake, N.Y.
JOHN W. BRANNAN, M.D., New York.
JUDSON DALAND, M.D., Philadelphia.
CHARLES L. MINOR, M.D., Asheville, N.C.

* Deceased.

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LIST OF OFFICERS.

Presidents.

NAME	YEAR
A. L. Loomis	1884–5
William Pepper	1886
Frank Donaldson	1887
A. L. Loomis	1888
V. Y. Bowditch	1889
Charles Denison	1890
F. I. Knight	1891
W. E. Ford	1892
R. G. Curtin	1893
A. H. Smith	1894
S. E. Solly	1895
J. B. Walker	1896
E. Fletcher Ingals	1897
E. O. Otis	1898
Beverley Robinson	1899
Abraham Jacobi	1900
Robert H. Babcock	1901
Samuel A. Fisk	1902
Norman Bridge	1903
James C. Wilson	1904
W. F. R. Phillips	1905
E. L. Shurly	1906
Thomas Darlington	1907
Thomas D. Coleman	1908
Charles E. Quimby	1909
Edward R. Baldwin	1910
John Winters Brannan	1911
Alexander D. Blackader	1912
Charles L. Minor	1913
James M. Anders	1914

Vice-Presidents.

NAME	YEAR
F. I. Knight, W. H. Geddings	1884-5
Frank Donaldson, Beverley Robinson	1886
V. Y. Bowditch, R. G. Curtin	1887
A. Y. P. Garnett, J. T. Whittaker	1888
J. R. Leaming, E. T. Bruen	1889
A. L. Gihon, H. B. Baker	1890
E. L. Trudeau, T. S. Hopkins	1891
E. Fletcher Ingals, Beverley Robinson	1892
A. H. Smith, E. O. Otis	1893
I. Hull Platt, E. L. Trudeau	1894
John H. Musser, G. R. Butler	1895
Charles E. Quimby, James A. Hart	1896
S. A. Fisk, John C. Munro	1897
Beverley Robinson, C. F. McGahan	1898
James A. Hart, R. C. Newton	1899
R. H. Babcock, J. W. Brannan	1900
Albert C. Peale, S. W. Langmaid	1901
Norman Bridge, W. F. R. Phillips	1902
James C. Wilson, H. S. Orme	1903
Thomas Darlington, Thomas D. Coleman	1904
S. G. Bonney, S. D. Risley	1905
A. D. Blackader, Henry Sewall	1906
Frank Fremont-Smith, C. L. Minor	1907
Judson Daland, Charles Fox Gardiner	1908
E. R. Baldwin, C. E. Edson	1909
James M. Anders, H. Longstreet Taylor	1910
Will Howard Swan, John H. Lowman	1911
Herbert Maxon King, Carroll E. Edson	1912
James M. Anders, C. D. Alton	1913
Lawrason Brown, Will Howard Swan	1914

Secretaries and Treasurers.

James B. Walker	1884–95
Guy Hinsdale	1895

LIST OF MEMBERS.

Honorary Members.

ELECTED

1888. Abbot, Griffith E., Leominster, Mass.
1887. Fisk, Samuel A., Brimfield, Mass.
1902. McBride, James H., Pasadena, Cal.
O. M. Reed, Boardman, Alhambra, Cal.
O. M. Robinson, Beverley, 42, West 37th Street, New York.
O. M. Schauffler, Edward W., Kansas City, Missouri.
1911. Stupart, Prof. R. F., Director, Dominion Meteorological Service, Toronto, Canada.
1885. Trudeau, Edward L., Saranac Lake, New York.
O. M. Tyndale, J. Hilgard, Richards Block, Lincoln, Nebraska.
1897. Weber, Sir Hermann, 10, Grosvenor Street, W., London, England.

Corresponding Members.

1912. Carpenter, Ford A., Weather Bureau, Los Angeles, Cal.
1898. Eyre, G. G., Claremont, Cape Town, South Africa.
1910. Forbes, Mr. N. Hay, F.R.C.S., Church Stretton, England.
1911. Grenfell, Wilfred, C.M.G., St. Anthony, Newfoundland.
1910. Gros, E. L., 23 Ave. du Bois de Boulogne, Paris, France.
1912. Henderson, Yandell, 410, Prospect Street, New Haven, Conn.
1910. Lamb, George, Indian Medical Service, c/o Medical Department, Royal Army, London.
1908. Liston, W. Glen, D.P.B., Capt., Indian Medical Service, Parel, Bombay Bacteriological Laboratory, Bombay, India.
1910. Rogers, Leonard, I.M.S., F.R.C.S., School of Medicine, Calcutta, India.

O. M., Original Member.

ELECTED

1904. Sandwith, Fleming Mant, 31, Cavendish Square, W., London.
1898. Sunderland, Septimus, 11, Cavendish Place, Cavendish Square, W., London.
1902. Weber, F. Parkes, 19, Harley Street, W., London.
1907. Wellman, F. Creighton, Tulane University, 1551, Canal Street, New Orleans, La.
1907. Williams, Leonard, 123, Harley Street, W., London.

Active Members.

1897. Alton, Charles D., 1310, Asylum Avenue, Hartford, Conn.
1899. Anders, James M., 1605, Walnut Street, Philadelphia.
1890. Anderson, B. P., 106, N. Cascade Avenue, Colorado Springs, Col.
1900. Arnold, Horace D., 520, Commonwealth Avenue, Boston.

1893. Babcock, R. H., 44, Bellevue Pl., Chicago.
1898. Baldwin, Edward R., Saranac Lake, N.Y.
1901. Barlow, W. Jarvis, Security Building, Los Angeles, Cal.
1907. Barnes, Harry Lee, Wallum Lake, R.I.
1902. Bergtold, William H., 1159, Race Street, Denver, Col.
1906. Billings, John S., jun., 149, Centre Street, New York City.
1897. Blackader, Alexander D., 236, Mountain Street, Montreal, Canada.
1895. Boardman, W. S., 63, Mt. Vernon Street, Boston.
1897. Bonney, S. G., Stedman Building, Denver, Col.
1885. Bowditch, V. Y., 506, Beacon Street, Boston.
1901. Bracken, Henry Martyn, 1010, Fourth Street, S.E., Minneapolis, Minn.
1891. Brannan, John W., 11, West 12th Street, New York City.
1894. Bridge, Norman, Temple Auditorium, Los Angeles, Cal.
1903. Brown, Lawrason, 104, Main Street, Saranac Lake, N.Y.
1903. Brown, Philip King, 350, Post Street, San Francisco, Cal.
1897. Brown, Sanger, Reliance Building, Chicago.
1907. Browning, Charles C., Story Bldg., Los Angeles, Cal.
1909. Bushnell, George E., U.S.A., Fort Bayard, New Mexico.
1886. Butler, G. R., 226, Gates Avenue, Brooklyn.

ELECTED

1896. CAMPBELL, W. A., 424, N. Nevada Ave., Colorado Springs, Col.
1910. CARRINGTON, P. M., U.S.M.H. and P.H. Service, St. Louis, Mo.
1898. CASSELBERRY, W. E., 15, East Washington Street, Chicago, Ill.
1906. CLAYTOR, THOMAS A., 1826, R. Street, N.W., Washington, D.C.
1901. COBB, J. O., U.S.M.H.S., Marine Hospital, Chicago.
1894. COLEMAN, THOMAS D., 505, Greene Street, Augusta, Ga.
1901. COLLINS, CHARLES FARNHAM, 50, West 55th Street, New York City.
1889. COOLIDGE, A., jun., 613, Beacon Street, Boston.
1902. CRAIG, R. W., Phœnix, Arizona.
1910. CRANE, BAYARD T., Rutland, Mass.

1907. DA COSTA, JOHN C., jun., 264, South 15th Street, Philadelphia.
1892. DALAND, JUDSON, 317, South 18th Street, Philadelphia.
1890. DARLINGTON, THOMAS, 27, Washington Square North, New York City.
1907. DARNALL, WM. EDGAR, 1704, Pacific Ave., Atlantic City.
1897. DAVIS, N. S., 7, West Madison Street, Chicago.
1910. DIXON, SAMUEL G., Harrisburg, Penn.
1905. DUNN, WILLIAM LEROY, Asheville, N.C.

1897. EDSON, CARROLL E., McPhee Building, Denver, Col.
1903. EDWARDS, WILLIAM A., Security Building, Los Angeles, Cal.
1903. ELLIOTT, J. H., 11, Spadina Road, Toronto, Canada.
1892. ELSNER, H. L., Fayette Park, Syracuse, N.Y.

1912. FARRAND, LIVINGSTON, 105, East 22nd Street, New York.
1910. FLOYD, CLEAVELAND, 1398, Beacon Street, Boston, Mass.
1885. FORD, WILLIS E., 266, Genesee Street, Utica, N.Y.
1911. FULTON, FRANK TAYLOR, 36, Prospect Street, Providence, R.I.

1896. GARDINER, C. FOX, 818, North Cascade Avenue, Colorado Springs, Col.
1886. GARNETT, A. S., Hot Springs, Ark.

ELECTED

1892. Gibson, William M., 260, Genesee Street, Utica, N.Y.
1909. Griffin, Walter Alden, Sharon, Mass.

1907. Hall, J. N., 452, Metropolitan Building, Denver, Col.
1912. Hamman, Louis V., 714, Park Avenue, Baltimore.
1893. Hance, I. H., Lakewood, N.J.
1891. Hart, James A., P.O. Box 144, Geneva, N.Y.
1900. Harvey, Thomas W., 463, Main Street, Orange, N.J.
1912. Hastings, Thomas W., 172, East 71st Street, New York.
1912. Hatfield, Charles J., 2008, Walnut Street, Philadelphia.
1896. Heffron, John L., 582, South Salina Street, Syracuse, N.Y.
1912. Hewlett, A. Walter, 1835, Cambridge Road, Ann Arbor, Michigan.
1893. Hinsdale, Guy, Hot Springs, Va.
1902. Hoagland, Henry W., 818, North Nevada Avenue, Colorado Springs, Col.
1905. Huddleston, John H., 145, West 78th Street, New York.

O. M. Ingals, E. Fletcher, Monroe Buildings, 104, Michigan Avenue, Chicago.

1889. Jacobi, A., 19, East 47th Street, New York.
1888. Jayne, W. A., 416, McPhee Building, Denver, Col.
1901. Jennings, Charles Godwin, 457, Jefferson Street, Detroit, Michigan.

1910. Kahlo, George D., White Sulphur Springs, West Virginia.
1904. King, Herbert Maxon, Liberty, New York.
1907. Kinghorn, Hugh M., 14, Church Street, Saranac Lake, N.Y.
1899. Klebs, Arnold C., care of J. M. Forbes and Co., Sears Bldg., Boston.
1903. Kyle, D. Braden, 1517, Walnut Street, Philadelphia.

1910. Landis, H. R. M., 11, South 21st Street, Philadelphia.
1899. Le Fevre, Egbert, 40, West 72nd Street, New York.
1909. Locke, Edwin Allen, 311, Beacon Street, Boston, Mass.
1913. Lord, Frederick T., 305, Beacon Street, Boston.

ELECTED

1904. LOWMAN, JOHN H., 1807, Prospect Ave., S.E., Cleveland, O.
1907. LYMAN, DAVID RUSSELL, Wallingford, Conn.

1906. MANGES, MORRIS, 72, East 79th Street, New York.
1910. MARCY, ALEXANDER, Riverton, N.J.
1902. MARVEL, PHILIP, 1616, Pacific Avenue, Atlantic City, N.J.
1887. MAYS, THOMAS J., 1829, Spruce Street, Philadelphia.
1905. MILLER, JAMES ALEXANDER, 18, West 51st Street, New York.
1909. MINER, CHARLES H., 115, South Franklin Street, Wilkesbarre, Pa.
1909. MINER, STANLEY G., 58, Cadillac Square, Detroit, Mich.
1899. MINOR, CHARLES L., Asheville, N.C.
1909. MORGAN, JAMES DUDLEY, 919, Fifteenth Street, N.W., Washington, D.C.

1895. NEWTON, R. C., 42, Church Street, Montclair, N.J.
1907. NICHOLS, ESTES, 655, Congress Street, Portland, Maine.
1907. NICHOLS, JOHN B., 1321, Rhode Island Ave., N.W., Washington, D.C.
1912. NORRIS, GEORGE W., 1530, Locust Street, Philadelphia.

1888. OTIS, E. O., 381, Beacon Street, Boston.

1913. PARFITT, C. D., Gravenhurst, Ontario, Canada.
1912. PATERSON, ROBERT CHILDS, Ste. Agathe des Monts, Province of Quebec, Canada.
1887. PEALE, A. C., National Museum, Washington, D.C.
1906. PERKINS, JAY, 106, Waterman Street, Providence, R.I.
1895. PHILLIPS, W. F. R., Mobile, Alabama.
1887. PLATT, WALTER B., 802, Cathedral Street, Baltimore.
1902. POTTENGER, F. M., 1100, Title Insurance Building, Los Angeles, Cal.
1905. PRATT, JOSEPH H., 317, Marlborough Street, Boston.
1905. PRYOR, JOHN H., 26, Linwood Ave., Buffalo, New York.

1891. QUIMBY, CHARLES E., 278, West 86th Street, New York.

1885. RICE, C. C., 123, East 19th Street, New York.
1901. RICHARDSON, CHARLES W., 1317, Connecticut Avenue, Washington, D.C.
1904. RICHER, A. J., Ste. Agathe des Monts, Province of Quebec, Canada.

ELECTED

1890. Robinson, W. D., 2012, Mount Vernon St., Philadelphia.
1902. Rochester, Delancey, 469, Franklin Street, Buffalo, N.Y.
1892. Roe, John O., 44, South Clinton Street, Rochester, N.Y.
1890. Rogers, E. J. A., 222, Colfax Avenue, Denver, Col.

1905. Schauffler, William Gray, 400 Madison Ave., Lakewood, N.J.
1901. Sewall, Henry, 433-4, Majestic Building, Denver, Col.
1910. Shurly, B. R., 32, Adams Avenue W., Detroit, Mich.
1890. Smith, A. Alexander, 18, West 51st Street, New York.
1887. Smith, Frank Fremont, 1808, Massachusetts Ave., Washington, D.C.
1911. Steiner, Walter R., 4, Trinity Street, Hartford, Conn.
1900. Stevens, Martin L., Asheville, N.C.
1904. Stone, Arthur K., 543, Boylston Street, Boston, Mass.
1910. Swan, John M., 457, Park Avenue, Rochester, New York.
1901. Swan, Will Howard, 1440, North Nevada Avenue, Colorado Springs, Col.

1892. Taylor, H. Longstreet, 75, Lowry Arcade, St. Paul, Minn.
1907. Taylor, J. Gurney, 514, Goldsmith Building, Milwaukee, Wisconsin.
1896. Taylor, J. Madison, 1504, Pine Street, Philadelphia.
1912. Thayer, W. S., 406, Cathedral Street, Baltimore.
1910. Thomas, John D., 1726, M. Street, N.W., Washington.

1897. Whitcomb, H. H., 622, Dekalb Street, Norristown, Pa.
1911. White, William Charles, Bedford Ave. and Wandless St., Pittsburg, Pa.
1898. Whitney, Herbert B., 320, Temple Court, Denver, Col.
1898. Williams, Harold, 528, Beacon Street, Boston.
1885. Williams, H. F., 416, Grand Avenue, Brooklyn.
1911. Williams, Linsly R., 882, Park Avenue, New York City.
1910. Wilson, Gordon, 1315, North Charles Street, Baltimore.
O. M. Wilson, James C., 1509, Walnut Street, Philadelphia.
1913. Wood, Nathaniel K., 259, Beacon Street, Boston.

Total, 136 active members.

MINUTES.

The Thirtieth Annual Meeting of the Association was held in the New Willard Hotel, Washington, D.C., on May 6, 7, and 8, Dr. Charles L. Minor, the President, in the chair. The following were present during the meeting :—

Dr. C. D. Alton,
Dr. J. M. Anders,
Dr. H. D. Arnold,
Dr. R. H. Babcock,
Dr. E. R. Baldwin,
Dr. H. L. Barnes,
Dr. A. D. Blackader,
Dr. V. Y. Bowditch,
Dr. H. M. Bracken,
Dr. J. W. Brannan,
Dr. Lawrason Brown,
Dr. P. K. Brown,
Dr. C. C. Browning,
Dr. G. E. Bushnell,
Dr. P. M. Carrington,
Dr. W. E. Casselberry,
Dr. T. A. Claytor,
Dr. T. D. Coleman,
Dr. J. Daland,
Dr. T. Darlington,
Dr. W. E. Darnell,
Dr. W. L. Dunn,
Dr. J. H. Elliott,
Dr. H. L. Elsner,
Dr. L. Farrand,
Dr. C. Floyd,
Dr. F. T. Fulton,
Dr. A. C. Getchell,
Dr. W. A. Griffin,
Dr. J. N. Hall,
Dr. C. J. Hatfield,
Dr. G. Hinsdale,
Dr. J. H. Huddleston,
Dr. E. F. Ingals,
Dr. A. Jacobi,
Dr. C. G. Jennings,
Dr. G. D. Kahlo,
Dr. H. M. Kinghorn,
Dr. D. B. Kyle,
Dr. H. R. M. Landis,
Dr. J. H. Lowman,
Dr. D. R. Lyman,
Dr. A. Marcy,
Dr. P. Marvel,
Dr. J. A. Miller,
Dr. C. H. Miner,
Dr. C. L. Minor,
Dr. E. Nichols,
Dr. J. B. Nichols,
Dr. G. W. Norris,
Dr. E. O. Otis,
Dr. A. C. Peale,

Dr. Jay Perkins.
Dr. W. B. Platt,
Dr. F. M. Pottenger,
Dr. J. H. Pratt,
Dr. C. W. Richardson,
Dr. B. Robinson,
Dr. W. D. Robinson,
Dr. De L. Rochester,
Dr. H. Sewall,
Dr. B. R. Shurly,
Dr. Fremont Smith,
Dr. W. R. Steiner,
Dr. M. L. Stevens,
Dr. A. K. Stone,
Dr. J. M. Swan,
Dr. W. H. Swan,
Dr. J. M. Taylor,
Dr. W. S. Thayer,
Dr. J. D. Thomas,
Dr. H. H. Whitcomb,
Dr. Wm. Ch. White,
Dr. Linsly R. Williams,
Dr. Gordon Wilson,
Dr. J. C. Wilson.

Dr. James Dudley Morgan and Dr. Thomas W. Hastings sent papers, but were unavoidably detained. Drs. S. G. Bonney, N. S. Davis, B. T. Crane, C. E. Edson, J. H. Hance, J. A. Hart, W. A. Jayne, A. C. Klebs, E. A. Locke, and W. G. Schauffler sent excuses.

Besides the President's address, the scientific programme included twenty-four papers, which were presented and discussed in three sessions.

Secretary and Treasurer's Report.

The Association at the completion of its thirtieth year has 10 honorary members, 16 corresponding members, and 135 active members. We have lost two by death. Dr. Roland G. Curtin, who joined in the first year, died at his home in Philadelphia on March 14, 1913. He was devoted to this Association and to its individual members. We have all suffered a personal loss, and shall always revere his memory. Dr. Anders will read a memorial of him later in the meeting.* The second loss by death was that of our honorary member, Dr. C. Theodore Williams, of London. He was elected in 1897, and died December 15, 1912, in the 75th year of his age. He was President of the Medical Society of London in 1889, and was five times elected President of the Royal Meteorological Society. He was deeply interested in America, and was the author of papers on "The High Altitudes of Colorado and their Climates"; "The Climate of Southern California"; "Meteorology in relation to Hygiene"; "Aerotherapeutics in Lung

* See page xxiii.

Diseases," and other works, which showed his practical knowledge of climate and his high appreciation of American climates and resorts in treating pulmonary disease. He took a great interest in building the Brompton Sanatorium at Frimley, and was to the end Treasurer of the Queen Alexandra Sanatorium at Davos, Switzerland.

The Council has dropped one member for non-payment of dues.

The last volume of the Transactions of the Association has been printed as usual in London, and contains more papers than any previous volume. It has been distributed to various libraries and journals as in the past.

The Council has considered six names presented at the last meeting, and recommends five for election to active membership. The Council at the meeting in January recommended the election of Dr. S. W. Langmaid, of Boston, to the Honorary List. Dr. Langmaid has been a member of good standing for twenty-six years.

Several communications have been received by the Secretary, which should be brought before the Association. Dr. A. W. Keen, of Philadelphia, has called our attention to the proposed memorials to Lord Lister, in which we are asked to participate. There will be an international memorial fund, from which awards will be given for the most notable contributions to surgery in any part of the world; there will also be a memorial tablet and a monument.

There will be held at London, August, 1913, the Seventeenth International Congress of Medicine, and we should send delegates. Four of our members, Dr. Blackader, Dr. Manges, Dr. Elsner, and Dr. Edson have signified their intention of attending, and, if agreeable to the Society, would be willing to serve as delegates.

We have also been invited to participate in the Ninth International Congress of Hydrology, Climatology, and Geology at Madrid, October 15-22, 1913. If any members can attend, will they kindly send their names to the Secretary? This Congress will be an important one, and doubtless very interesting.

We have also been requested by Dr. Harry M. Halloch, the Medical Director of the Arkansas Hot Springs Reservation, to urge the passage of a Bill now pending in Congress for determining the physiological and therapeutic effects of

the hot waters from these springs, and report on their use in the cure of disease. This plan should have our active support, as it will be conducted, if authorized, by the United States Government, which is endeavouring to put our knowledge of mineral waters on a truly scientific basis.

FINANCIAL STATEMENT.

June 1, 1912. Balance in hand	$163.18
Received from dues	872.00
Total	1,035.18
Expenses, as per Treasurer's books	490.73
Balance in hand, May 1, 1913	$544.45

GENTLEMEN,—At the Montreal meeting the Chairman of this Committee had the honour of reading a paper on a system of notation for recording physical findings in pulmonary disease. As a result of his discussion on that paper, it was moved that a committee to recommend some system of notation for this purpose be appointed, and Dr. Blackader, then President, appointed your Committee. Being unable to report at the Hartford meeting, it begs leave now to submit its report.

Your Committee has no doubt that it will be most desirable, and will conduce to more careful and accurate work, and to the ease of interchange of medical facts between physicians if a uniform system of signs could be adopted by the members of this Association, and by the medical profession of the country generally. When, however, we come to consider ways and means of accomplishing this purpose it becomes evident that there are several things which make the success of such a project most problematic. The devising of such a system seems by far the least difficult part of the question. Many such questions, some of them most excellent, many most complex and difficult, have been proposed. We need only recall that two such distinguished physicians as Drs. Sahli and John H. Musser, the latter an honoured member of this Society, have published excellent schemes which yet wait for general adoption.

The Chairman of your Committee has for a number of years now used with great satisfaction a system of his own, and which he believes to be practical, simple, clear, and easy

of application, and he would be unwilling to get along without the assistance which it gives him. But granted that this Society prepare such a system and lends it the support of its honoured name, and even that it were of extreme excellence, anyone familiar with past experiences in this matter and knowing human nature will recognize that only a few will take the trouble to adopt it and be willing to adhere to it closely and unmodified. Yet as soon as it is modified to suit the wishes of each man, then while it may be useful to him individually, yet as a means of common record and communication of scientific observations between various medical workers it loses all value. Indeed, the history of the various systems that have from time to time been proposed shows this very clearly. No one, whatever the distinction of his name, has been able to recommend a scheme that could win anything like a general recognition from the medical profession.

But there is another prerequisite to any satisfactory system which so far has not been fulfilled. We refer to the lack of any close uniformity in the medical nomenclature of the physical signs of pulmonary disease. Unless there is such a uniformity it is useless to talk of the general use of any system of notation, yet a study of the various American text-books of physical diagnosis shows the greatest variation in this nomenclature, so that reports of physical findings are by no means easy to compare.

In different parts of the world, even in different parts of the same country, not to mention different parts of the same town, different physicians use widely different terms to connote the same conditions, and use many and varied terms to describe one and the same sound.

Indeed, your Committee feels that as a prerequisite to the task given them would be the work of a national, or better, an international committee to unify the medical nomenclature of the physical signs of pulmonary disease. Therefore, your Committee believes that while such systems can be very useful to the men who make and use them systematically and enable them to record their findings more quickly, clearly, and accurately, and render these records more simple and easy of reference, and while a simple system would be valuable generally if it could be adopted, owing to the reasons already given the recommendation of such a system

does not seem to us to be practical, and hence we consider it inadvisable that the Society should at this time recommend or try to introduce a definite system of signs for general adoption.

CHARLES L. MINOR, *Chairman*,
LAWRASON BROWN,
J. H. ELLIOTT,
W. L. DUNN,
J. H. PRATT,
W. A. GRIFFIN,
T. D. COLEMAN.

MEMORIAL.

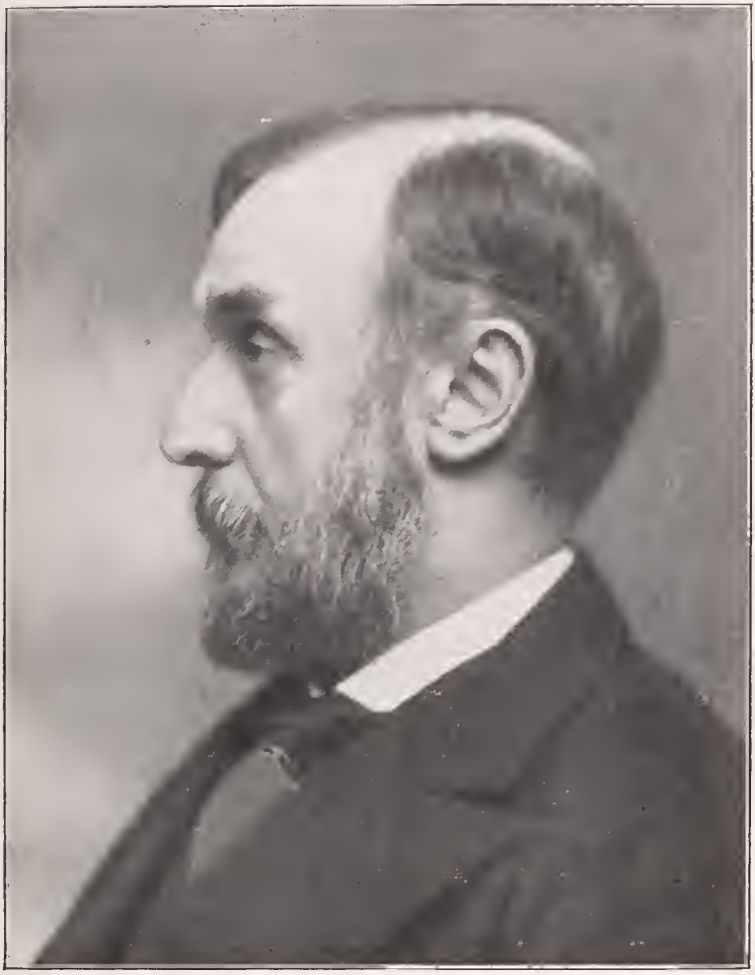
CHARLES THEODORE WILLIAMS.

The Association regrets to have to announce the loss of one of its honorary members, Charles Theodore Williams, who died on December 15, 1912. "Charles Theodore Williams was born in 1838, and was the son of the late Dr. C. J. B. Williams, F.R.S. He received his education at Harrow, and subsequently at Pembroke College, Oxford, where he graduated with honours in Natural Science. He was a student at St. George's Hospital, where he became demonstrator of anatomy and physiology; he also studied in Paris. He became M.D. in 1869, and was made a Fellow of the Royal College of Physicians in 1871. He was elected Councillor in 1891, and Censor in 1899. He delivered the Lumleian Lectures in 1893, choosing as his subject 'Aerotherapeutics in Lung Diseases,' and was Harveian lecturer in 1911, his discourse being entitled 'Old and New Views on the Treatment of Consumption.' At the College of Physicians of London, in addition to his academic position, Dr. Williams was well known as an excellent organizer, and his aid was always sought in the various social gatherings occasionally given by the College. He was also a benefactor of the College, having given £1,000 to the Endowment Fund, and being instrumental in founding the Bisset-Hawkins memorial medal. Dr. Williams held a prominent position in the Medical Society of London, having occupied the presidential chair in 1889. He also delivered the Lettsomian Lectures in 1876, and the annual oration in 1884. He likewise took much interest in the Meteorological Society, of which he was the Honorary Treasurer and six times President.

"The Medical Graduates College and Polyclinic, of which he was President, owes much to his energy and perseverance. At his college at Oxford he founded four university and two college scholarships in human anatomy, physiology, and pathology, including bacteriology in relation to medicine; he was elected an honorary Fellow of his college in 1907.

"This record of good and self-sacrificing work shows how general as well as how deep Dr. Williams's interest in the welfare of the medical profession was, but his name has always been associated with the Brompton Hospital for Consumption and Diseases of the Chest. The hospital was founded in 1841, and Dr. C. J. B. Williams was Consulting Physician from 1842 to 1889. Dr. Theodore Williams was appointed Assistant Physician in 1867, Physician in 1871, and Consulting Physician in 1894. His devotion to the interests of the hospital was recognized by the Committee of Management and by his colleagues, and nowhere will his loss be more sincerely felt than at this institution. He did not hold an appointment at a general hospital, and all his energies were directed to promoting the interests of the Brompton Hospital. Dr. Williams's writings on diseases of the chest, more particularly pulmonary tuberculosis, are universally known, and they are a record of work done or experience gained at Brompton. His best known works were 'Pulmonary Consumption: its Modes of Arrest, Treatment, and Duration,' and 'Aerotherapeutics.' He also read several papers before the medical societies on similar topics, and his article on 'Treatment of Phthisis at High Altitudes,' at the Royal Medical and Chirurgical Society, attracted much attention. It was especially on the climatic treatment of pulmonary tuberculosis that Dr. Williams's writings were prominent. After a visit to Davos in 1869 he gave a thorough trial to the high altitude treatment, and he watched the effect on a large number of his patients, sending them not only to the mountain resorts of Switzerland, but also to North and South America and South Africa.

"When the sanatorium treatment was generally introduced into England Dr. Williams was again to the fore. He took a prominent part in the building of the King Edward VII Sanatorium, where he was subsequently appointed Consulting Physician, and for the assistance he had given towards



Dr. C. THEODORE WILLIAMS.

Born August 29, 1838. Died December 15, 1912.
Elected Honorary Member, 1897.

the arrangements for the erection and equipment of the sanatorium he was awarded the honour of M.V.O. He took a similar interest in the building of the Brompton Hospital Sanatorium at Frimley.

"Dr. Williams was Physician to the English and Scottish Law Life Assurance Office. He was one of the original members of the Life Assurance Medical Officers' Association, of which he was President in 1900-01. Apart from public considerations, his death will be greatly regretted by a large number of personal friends. His genial and kindly disposition was very attractive, and the numerous members of the medical profession who, through his long service at the Brompton Hospital, acted at his house physicians will always remember with pleasure the enthusiasm and interest which he threw into his work."—*The Lancet*, December 21, 1912.

ROLAND GIDEON CURTIN.—AN APPRECIATION.*

By J. M. ANDERS, M.D.

Roland Gideon Curtin, A.M., M.D., Ph.D., of distinguished lineage traceable back to a royal line, a native of Pennsylvania, was an honoured alumnus of the University of Pennsylvania whose recent passing away this Association truly mourns. Medical, fraternal, and scientific organizations and local institutions with which his name had been long and prominently linked all feel his loss, but no society more keenly than the American Climatological Association, and to this body, to us, the name of Curtin will ever be of revered and undying memory.

His deep, unfailing interest in this Association, the touch of his master-spirit in the conduct of its business affairs, as well as the evident tokens of high regard and fraternal love which he manifested for its members, are facts which are universally known and appreciated by his fellow-members.

A considerable portion of the membership of this

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* Read before the American Climatological Association, Washington, D.C., May 8, 1913.

Association remember his appearance at the flush of his vigorous manhood—a sturdy, large frame, erect carriage, deliberate gait, black hair and beard, massive, symmetrical head, dark and sympathetic eyes, radiant countenance, and frank, cordial manner. One who knew him long and intimately has well said, "Time worked the inevitable change for all things material, whitened his hair, sapped his vigour, and at last pierced him with pain, but could not embitter his heart, or steal from him his love or his friends."

Dr. Curtin's Celtic origin was shown in the shock of pleasant surprise which he constantly gave to his friends by his inimitable wit. Unlike many witty men, he also had humour, which sprang up exuberantly as from a fountain, and rolled on with evident delight to his hearers.

It has been left to another and distinguished member of this Association to present a comprehensive survey of the events and achievements of our deceased brother's long and useful life.

In this connection it should, however, be stated that an unmistakable criterion of his personal worth and professional services was beautifully manifested at a meeting of the Philadelphia Alumni Society of the University of Pennsylvania, held within one month of his death, in resolving to create the "Roland G. Curtin Scholarship for the Sons of Physicians," an honour the bestowal of which is almost unique in medical circles.

It is altogether noteworthy that in the unseemly rush for preferment, place, or power Dr. Curtin was ever conspicuous by an opposite tendency, and yet he was a man of executive ability, and distinct leadership in his chosen profession.

His gracious and kind presence, like his cheering words, attracted and inspired all who fell within his personal contact. Moreover, as the result of esteem paid to merit he was clothed with many professional honours, all of which he bore with becoming dignity and modesty.

Dr. Curtin's tastes and habits were simple and unostentatious and had it not been for the kind forethought and unselfish interest of one near and dear to him, he would most probably have failed to provide himself with the means necessary to make comfortable and to brighten the day of retirement, which came to him as the result of a disabling malady early in the declining period of his life.

ROLAND G. CURTIN, M.D.

President, 1893. Died March 14, 1913

True, Dr. Curtin enjoyed an extensive practice for a long term of years, both as family physician and a consultant, but, like the Christ, "he made himself of no reputation, and took upon him the form of a servant."

His earnings represented a large pecuniary consideration, but they were in large part laid out for the comfort and enjoyment of others, for his charitable acts were indeed many, although known to but few, and then only from necessity.

The spirit of commercialism that characterizes the merchant or the manufacturer, and unfortunately a considerable contingent of the medical profession even, found no place with him. For attendance on wealthy patients he would exact no more than the customary fee, while the deserving poor were privileged to come to him "as a friend."

Dr. Curtin believed that the best services of the profession should be obtainable by all classes in serious medical and surgical diseases, and he was known to respond to the calls of poor patients residing in the outlying districts of Philadelphia, while remunerative ones and those high in the social scale were obliged to wait. To his intimate friends it was definitely known that Dr. Curtin's services were rendered quite as freely and faithfully to the dependent as to the better-to-do classes. He thus combined industry and fidelity to duty with personal sacrifice in the discharge of his professional labours—in truth, combined professional with angelistic work.

Strength of will he had, and slow to experience a change of heart after he had reached a definite decision on any given subject he was, yet he ever maintained an open mind and a wholesome tolerance of the views of others. In no sense was Dr. Curtin a man of confined views or sentiments, but one of liberal, charitable, and enlarged sympathies. In every relationship of life, however, he never knowingly violated his sense of duty, nor surrendered his convictions of right.

Dr. Curtin was easily one of the leading physicians of his day and generation; he was an astute diagnostician, helpful and generous consultant, sympathizing by temperament and imagination with the past in medicine, particularly as regards therapeutics, still methinks it will be Curtin the man—a life foundationed, shielded, and panoplied by the highest ideals which it exemplified—that will endure without

perishing, and serve as a guiding star to succeeding generations of the medical profession.

His sympathies were ever warmly enlisted for the promotion of fraternity and unity among physicians, and also between physicians and undergraduate students. Many a sleeping conscience was aroused to a realization of the importance of bringing into brotherly sympathy with one another the members of our profession by his personal touch; he thus left to his survivors—to us—an example in this respect which deserves universal imitation.

It was the writer's good fortune to see him at intervals during his last protracted illness, and he was much impressed with certain rare features of his lofty character. The basic stratum of the peculiar qualities impressed by nature was easily a deep sense of responsibility to God, and this, like a holy ark, kept him safe amid the rising flood of temptations, scepticism, and intrigue that tends to engulf every professional life of mark.

No false steps to be retrieved, nothing realized that had not been previously conceived with care and caution, he marched under the escort of the god of fortune, his reputation unmarred by the shadow of the smallest cloud on his integrity.

Ever conforming to the highest principles of rectitude in conduct, ever a heart to do well, ever "kindly with his kind," he poured his life into his work and struggles, and won, fully realizing, although unconsciously, the cherished ideals of his profession.

Such a character properly understood can lead only to a highly favourable estimate of its merits; beautiful in itself, but more than this, capable of enhancing the beauty of its fellow-characters.

It is deservedly noteworthy that Dr. Curtin died on March 14, the forty-seventh anniversary of his graduation in medicine, and seven years to the day after the first of a series of attacks of recurrent pulmonary œdema, which culminated in his last fatal illness. He was buried on the ninth anniversary of Mrs. Curtin's death—for him the saddest day of the year during the declining period of his life. He was buried on St. Patrick's Day of the present year, which day his daughter, Miss Curtin, states he had always spent reminiscing of his father and Ireland.

Such was the strange coincidence of events that closed the life of one possessing the most tender susceptibilities, and one whose death was a calamity to the profession of which he was a chief ornament.

"Blessed are the dead who die in the Lord."

"Even so saith the spirit; for they rest from their labour, and their works do follow them."

ERNEST LORENZO SHURLY.

Doctor Ernest Lorenzo Shurly, of Detroit, physician, specialist, teacher, clinician, and public-spirited citizen, died of heart disease May 10, 1913, aged 67.

Dr. Shurly had become eminent in the medical profession not only of the middle west but of the nation as well. He was graduated from the medical department of the University of Buffalo in 1866, and had been engaged in private and Army practice before locating in Detroit, having been acting assistant surgeon in the United States Army, with service in Indian campaigns and in the Yellowstone Expedition. The doctor was a pioneer in the fight against tuberculosis, and through his instrumentality the first tuberculosis camp in Michigan was founded at Eloise, in Wayne County. He supervised research work on the lower animals for the purpose of studying tuberculosis, and published valuable contributions to scientific medicine on the results thereof. For years he had been connected with the teaching force of the Detroit College of Medicine, imparting instruction in diseases of the nose, throat, and chest. He was the founder of the department of laryngology in this institution, and an active lecturer and clinician until a few years ago, when he retired with the title of Emeritus Professor of this branch of medicine. For a number of years he was chief of staff of Harper, and member of the staff of Saint Mary's, Saint Luke's, and the Women's Hospital. He devoted considerable time to the problem of hospital management, and his executive ability was exemplified in marked degree as chief of staff at Harper.

Dr. Shurly's contributions to medical literature were various and valuable. He was not only a prolific writer for

the medical periodic press during his stringent life, but standard text-books bearing his authorship were likewise dedicated to medical science.

Although in recent years the doctor was not as eager to increase his practice as he was earlier in his medical career, he nevertheless maintained vital interest as an expert in his chosen speciality to the end, and realized the fulfilment of his often expressed desire that he might "die in the harness."

ERNEST L. SHURLY, M.D.

President 1906. Died May 10, 1913.

CONSTITUTION AND BYE-LAWS.

CONSTITUTION.

Article I.—Name.

This Society shall be known as the American Climatological Association.

Article II.—Object.

The object of this Association shall be the study of *Climatology and Hydrology and of Diseases of the Respiratory and Circulatory Organs.*

Article III.—Membership.

Section 1.—This Association shall consist of *active, corresponding,* and *honorary* members, the former not to exceed 150 and the latter not to exceed twenty (20).

Section 2.—Names of candidates for active membership, whose applications shall have been endorsed by *three* (3) active members, shall be sent to the Secretary at or before the annual meeting at the second business session of which they shall be read and then lie over until the next annual meeting, when such as are approved by the Council shall be balloted on. Three (3) black balls shall be sufficient to reject a candidate. The Council shall have power to nominate active members.

Section 3.—The power of nominating honorary and corresponding members shall be vested in the Council. The election shall be conducted in the same manner as that for active members. Honorary members shall enjoy all the privileges, but shall not be allowed to hold any office or cast any vote.

Section 4.—Any member of the Association absent from the meetings, in person or by contributed paper, for three (3) consecutive years, without sufficient cause, shall be dropped from the list of members by vote of the Council.

Article IV.—Officers.

Section 1.—The officers of this Association shall consist of a *President,* two *Vice-Presidents,* a *Secretary and Treasurer,* who, with five other members, shall constitute the *Council* of the Association.

Section 2.—*Nominations.* The officers, including the Council, shall be nominated by a committee of five (5) members, which committee shall be nominated by the President at the first session of each annual meeting and shall report at the business meeting.

Section 3.—*Elections.* The election of officers shall take place at the business meeting. A majority of votes cast shall constitute an election.

Section 4.—The President, Vice-Presidents, Secretary and Treasurer shall enter upon their duties at the close of the annual meeting at which they are elected, and shall hold office until the close of the next annual meeting, or until their successors are elected.

Section 5.—Members of the Council, other than the President. Vice-Presidents, Secretary and Treasurer, shall hold office for five (5) years.

Section 6.—*Vacancies.* Any vacancy occurring among the officers of the Association during the year may be filled by the Council.

Article V.—Duties of Officers.

President and Vice-Presidents.

The President and Vice-Presidents shall discharge the duties usually devolving upon such officers. The President shall be *ex officio* Chairman of the Council.

Secretary and Treasurer.

As Secretary, he shall attend and keep a record of all the meetings of the Association and of the Council, of which latter he shall be *ex-officio* Clerk. At each annual meeting he shall announce the names of all who have ceased to be members since the last report. He shall superintend the publication of the Transactions, under the direction of the Council. He shall notify candidates of their election to membership. He shall send a preliminary notification of the annual meeting two (2) months previous thereto, and the programme for the annual meeting at least two (2) weeks previous to its assembly, to all the members of the Association. He shall also send notification of the meetings of the Council to the members thereof. At each annual meeting of the Association he shall read the minutes of the previous meeting and of all the meetings of the Council that have been held during the current year.

As Treasurer, he shall receive all moneys due, and pay all

debts therewith. He shall render an account thereof at the annual meeting, at which time an auditing committee shall be appointed to report.

Article VI.—Council.

The Council shall meet as often as the interests of the Association may require.

Four (4) members shall constitute a quorum.

It shall have the management of the affairs of the Association, subject to the action of the Association at its annual meetings.

It shall consider the claims of candidates recommended to it for admission to membership.

It shall not have the power to make the Association liable for any debts exceeding in total one hundred dollars ($100), in the course of any one year, unless specially authorised by a vote of the Association.

It shall have the entire control of the publications of the Association, with the power to reject such papers or discussions as it may deem best.

It shall have power to nominate active members at the annual meeting.

The Council shall have power to invite any gentleman, not a member, to read a paper at the annual meeting, on any subject within the scope of the objects of this Association.

The Council shall determine questions by vote, or—if demanded—by ballot, the President having a casting vote.

The Council shall constitute a Board of Trial for all offences against the Constitution and Bye-laws, or for unbecoming conduct, and shall have the sole power of moving the expulsion of any member.

The President, or any two members, may call a meeting, notice of which will be transmitted to every member two (2) weeks previous to the meeting.

Article VII.—Papers.

Section 1.—The titles of all papers to be read at any annual meeting shall be forwarded to the Secretary not later than one (1) month before the first day of the meeting, in order to appear on the printed programme.

Section 2.—No paper shall be read before the Association which has already been printed or been read before another body.

Article VIII.—Quorum.

A quorum for business purposes shall be ten (10) members

Article IX.—Amendments.

This Constitution may be amended by a four-fifths ($\frac{4}{5}$) vote of all the members present at an annual meeting, provided that notice of the proposed amendment has been printed in the notification of the meeting at which the vote is to be taken.

BYE-LAWS.

(1) Meetings of the Association shall be held annually.

(2) The time and place of the meetings shall be determined by the Council.

(3) The dues of active members shall consist of an annual assessment not to exceed seven and a half ($7.50) dollars. Members in arrears shall not be entitled to vote. Those in arrears for two (2) years may be dropped from membership by recommendation of the Council.

(4) Order of business meeting.

First day:—

Calling the roll of members.
Minutes of previous meeting.
Secretary's and Treasurer's reports.
Appointment of auditing committee.
Appointment of nominating committee.
Report of Council on recommendations for membership.

Second day, Morning session:—

Reports of nominating committee and auditing committee.
Election of officers.
Election of members.
Report of committee on health resorts.
Miscellaneous business.
Adjournment of business meeting.

PRESIDENT'S ADDRESS.

A RETROSPECT AND A PROSPECT.

By CHARLES L. MINOR, M.D.
ASHEVILLE, N.C.

Gentlemen of the American Climatological Association,—To-day our Society celebrates its thirtieth birthday; allow me to be the first to congratulate you upon that auspicious fact.

Organized in 1883, among its founders were such leaders of the medicine of that day as Alfred Loomis, William Pepper, Frederick Shattuck, J. R. Leaming, D. N. Camman, John H. Musser, J. B. Walker and our late honoured friend, Roland Curtin, while Beverley Robinson, now an honorary member, and J. C. Wilson and E. Fletcher Ingals, the only two original members now left on the active list, are with us to-day. It was such men as these who set for the Society that standard of performance which it is our duty to maintain.

One whole generation has elapsed since that time, and, in so progressive a profession as ours, thirty years bring changes so great as to almost revolutionize it, through the development of new ideas and the opening up of new territories in the field of knowledge.

In those three decenniums the advance in our art has been

enormous, as great or rather greater and more momentous than in any similar period in medical history.

Bacteriology, then scarcely recognized, may be said to have had its birth, certainly to have begun its real development from the little-heard-of study of a few enthusiasts to the position of one of the chiefest foundation-stones of medical science.

Antisepsis and asepsis have revolutionized not only surgery, but also medicine.

The whole brilliant hypothesis of immunity has been formulated and from it have been evolved serum and vaccine therapy, which are conferring such inestimable blessings on suffering humanity.

Laboratory diagnosis has assumed an importance that the leaders of that day could not possibly have anticipated and has become the indispensable handmaid of the up-to-date physician.

Our whole knowledge of tuberculosis has been recast, and the pessimism of the profession of 1884, as to its curability, has been replaced by an intelligent optimism based on proven scientific facts.

Nothing could better show the advance in this last realm than the perusal of the Presidential Address of Dr. Loomis in 1885.

He, who, in that day in this country, was the leading authority on this disease, discusses whether phthisis has its origin in an infection, and remarks that "there has never perhaps been a period when there was so great uncertainty in the minds of the profession in regard to the ætiology or morbid anatomy of phthisis as at present. For one class of observers pulmonary phthisis is an inflammation of the pulmonary substance, which may or may not be complicated by tubercle. Another class maintain that the tubercle is the primary and essential lesion of all phthisis."

"Still more recently certain investigators maintain that there is a specific material which may or may not be accompanied by the histological element of tubercle, but which always has a specific form of bacillus as the sole exciting cause of its development." . . . "Koch's statement that the repeated entrance into healthy lungs of a small number of the specific bacilli of tuberculosis will cause chronic phthisis, and that the simultaneous admission of numerous bacilli will produce acute cases, is yet unproved."

And these were the words of a leader but a short thirty years ago! When such changes occur in so short a time it is evident that only by constant growth and by adaptation to new conditions arising constantly, can medical men and medical societies hope to keep abreast of the times.

As, then, this meeting marks the thirtieth milestone in our course, it has seemed to me that I might well devote the time of the Presidential Address, not to the consideration of a medical topic, for on such topics you are to hear many able papers, but rather to a review of our Past, that from it we may draw inspiration and suggestions for our Future, in short to a Retrospect and a Prospect, and for this subject I will now ask your attention.

When we consider the names of the leaders who in the past have been on the rolls of this Society; when we look through the papers in the twenty-eight volumes of our *Transactions;* when we consider the position which this Society has held among its sister associations; and when, finally, we note the influence it has had on the course of things medical in this country in the past thirty years, I think that we can be justly proud of our Association.

Its personnel in the past I have already sufficiently referred to, the names I have mentioned speak for themselves. Few of us probably have had the time to inform ourselves of the work our predecessors have done. If we

did, we should be surprised at the large amount of valuable material that has first seen the light here.

As you all realize, the study of tuberculosis has taken a large place in our work, and the future historian of the tuberculosis campaign in the United States cannot afford to ignore what has been done by the members of this Society. Ours was the first Society to pay special attention to the subject. Valuable papers on the climatic treatment, careful studies on the use of tuberculin, pioneer work on nomenclature and classification, now taken up by the National Association and by the Sanatorium Association, important papers on early diagnosis and on pneumothorax and many others which had a strong influence on the development of an interest in tuberculosis in this country, can there be found.

But, as some of our Presidents have remarked in the past, we are not, and should not be, chiefly a society for the study of tuberculosis, although so much excellent work in that line has been done by our members. Hence it was natural and right that this work should be taken up by a special society which could include laymen as well as physicians in its ranks, could study its sociological as well as its medical aspects, and which could collect funds and carry on a campaign of education throughout the country for which a medical society, pure and simple, has not the time, and which could devote all its attention to the one subject of tuberculosis, while the Climatological Association, though always taking a deep interest in the subject, remains free to follow other subjects as well.

Hence we can only feel glad and proud of the active part that many of our members took in the founding of the National Association for the Study and Prevention of Tuberculosis, in which they have been active and prominent from the start, without, I am glad to say, losing one whit of their interest in the Parent Society, as the Climatological Associa-

tion may be named, for, if we look over the list of officers and directors of the National Association, we shall very quickly see that with justice we can be so called.

Again, we have a right to feel proud of the admirable work on diseases of the lungs and heart which has been done here, and especially on the physical diagnosis of those diseases, on which subject I think no society in this country has done more or better work. Our *Transactions* are an index of the more important advances in this subject and are a credit indeed to the Society which publishes them.

Naturally, climatology has had much attention devoted to it, though I notice that in recent years interest in this subject has fallen off considerably and much more work has been presented to us on more general subjects.

The important discussion of the relations between climatic and home treatment began with us and was carried further in the National Association.

Reports on many American climatic resorts have been read here. Two of our most devoted members have written the two best books in this country on "Climatology," and efforts have been made to encourage the teaching of climatology in our medical schools.

Again, our various mineral springs have been carefully studied and much information as to their qualities has been collected, though we are far from having on this subject the full information that is obtainable about the various European hydrotherapeutic resorts; and there is a movement on foot in Congress now, for which our support is requested, to have the springs of the United States carefully analysed and studied.

In these various ways, therefore, by its membership, by its standing and by its contributions to science, this Association has filled a valuable and important place in the medical annals of our country, while, as we all know, it has also the

well-deserved reputation of the most enjoyable, the most social, and the most closely-united society in our land.

And who can wonder at it, when we recall such genial souls as Knight and Walker, Curtin and Solly, Darlington, Shurly and Coleman, and a host of others, to meet whom always warms the cockles of our hearts?

So much, gentlemen, for a brief retrospect of the past of our Association. But if the future is to justify that past we cannot let our pride in the excellence of our Society make us forget that, like everything else of human origin, it must have, and does have, its faults.

If, then, we desire in the future, as in the past, to hold an honourable place among American medical societies, it behoves us critically to study present conditions in order to see what we can do to advance our Association, and to keep it in the forefront of progress.

In the past certain of our Presidents, Jacobi in 1900, Babcock in 1901, Bridge in 1903, have, in their addresses, struck this note of warning and of advice.

Dr. Jacobi, than whom there is no wiser counsellor, said: "It is hardly necessary to point to or emphasize the established policy of this Association, to admit only men whose positions have been established and who, through at least a few publications connected with our study, have proved their right to apply."

Dr. Babcock, in 1901, said: "Let us not as an Association, however, devote our energies too exclusively to the climatology of consumption, ignoring the other natural means of cure in the treatment of diseased conditions."

Dr. Bridge said, in 1903: "We cannot let the good fellowship which belongs to so harmonious a Society as ours, betray us into dropping the serious work of the Association. It seems to me that we are, through the character of our contract, under greater temptations to do this than any other of the societies of this Congress of Science."

Dr. Darlington, Dr. Coleman and others have dwelt on the fact that our name and constitutional limitations restrict us in letter, but should not restrict us in spirit, and by implication they both urged us to widen the scope of our work.

Many of the members have plainly recognized for a number of years that, partly from the limitation placed upon us by our name and our constitution, and partly because of the well-known social charm of our Society, we needed to watch with all care its development if we were to avoid getting into a rut or standing still instead of progressing as we must.

In the life of every organization there come critical periods pregnant with good or ill according as they are wisely met or blindly neglected. The present is, I believe, such a period for us, and it will be wise for us to-day to consider what we can do to meet to the fullest the demands of this rushing and eager twentieth century.

I would not for a moment have any of you suppose, because I would critically study the conditions of our Society, that I am lacking in loyalty to our Association. I have been devoted to it and proud of it ever since I had the honour of entering its ranks, and it is not disloyalty, I assure you, but rather the best of loyalty in one who loves it to consider if, and how, it can be improved.

Only in growth, gentlemen, is life; in lack of motion is death; he who stands still goes backward, and we must grow and expand, and improve, and keep up with the spirit of the times if we are to keep bright the honourable reputation handed down to us by the founders and earlier members of the American Climatological Association.

It was originally founded for the study of climatology, but even in the earlier Presidential Addresses it is evident that this seemed to be too narrow a field and diseases of the lungs and heart were soon added to it, and later hydrology.

But, gentlemen, times change and men change with them, and climatology, even with the powerful addition of diseases of the lungs and heart, no longer, I believe, offers a sufficient field for the activities of the Society.

Climatology does not awaken much real interest in the minds of the large majority of active medical men to-day, and for several years now there has been a growing feeling among our members that if we are to continue to grow and prosper and not to be merely a charming club of good fellows, but an active, scientific association doing valuable work in medical progress, we must widen our borders, must let it be understood that climatology is not the chief centre of our interest, must remove the restrictions set upon us by our name and by a clause in our constitution, and must feel ourselves free to study all subjects within the realm of clinical medicine.

Only so can we hope to keep up with the march of modern medical progress; only so draw to ourselves each year the best of those eager workers who are growing up everywhere in our country and with whom lies the future; only so can we keep up the honourable reputation and position which is ours; only so can we make our future worthy of our past.

The time is propitious! The demand to be met is clear, and if we will but have the courage to meet it wisely we need not fear for our future.

The popularity of laboratory work has brought into the programmes of many societies an excess of papers on purely laboratory topics of an extremely technical nature and which, however important, are nevertheless of subordinate interest compared to those in the great realm of practical internal medicine, and there is a distinct demand for more papers on clinical subjects, papers based on bedside observation; and while we all realize fully the importance of laboratory

work, and would not belittle it, we realize that the society which goes in for a large amount of this is apt to lose in practical medical interest.

There is, therefore, a very real demand for a society strictly clinical in its aims and scope and where all men interested in general clinical medicine, of which please remember climatology is a part, though a very small part, can bring their problems for discussion.

With the unquestionably waning interest in climatology as a medical subject and its relegation in some degree to the charge of the meteorologists, the name climatological no longer excites the interest of the new generation of medical workers and our Society has unquestionably suffered thereby, though fortunately the inclusion of diseases of the lungs and heart, which are so large and important a part of clinical medicine, has saved us from the asthenia which would have struck us had we been tied to climatology and hydrology alone; although the name of the Society does not indicate this opportunity.

But, gentlemen, it cannot continue in this way much longer without harm, and the time has come when we must widen our scope or suffer the consequences which always come to those who fail to comprehend in time the needs and demands of the age.

That we have a wonderfully choice membership of fine fellows, that it is a very real and personal pleasure to us to meet each other from year to year, that the friendships engendered here are deeper and stronger than in any other society of which I know, is not enough.

That we have always had, and now have, many distinguished names upon our rolls, that there have been read before us in the past many valuable communications, is not in itself enough, unless we can make our Society so attractive and desirable by its broad and catholic scope that the best

of the younger men, who, mind you, gentlemen, will be the distinguished men of the next fifteen or twenty years, shall feel it a privilege, an honour and a benefit to enter our ranks. Unless they do so we must inevitably suffer, for it is from such men that our ranks must be fed. They must realize that not good fellowship alone, not the recommendation of some kind friend alone, however honoured here, but in the last analysis the ability and willingness to do first-class clinical work and to read here strong papers, will be necessary for entrance to our ranks.

One of our most honoured members, Dr. Babcock, two years ago suggested widening the limitations set on papers to be read in this Society.

Other Presidents have neglected the letter of the law on these subjects, to our great benefit, and I myself, as President, this year took the doubtless unconstitutional liberty of asking for papers on subjects in the realm of general clinical medicine which, under the widest interpretation, do not come within the hard and fast restrictions with which we have bound ourselves.

Gentlemen, the opportunity is ours!

With the reputation this Society enjoys, with the distinguished names which honour its membership, with the great demand for a general clinical society, it is entirely within our power to open up a new era of prosperity; yes, much more, of usefulness, such as can scarcely be surpassed in our past history, honourable though it has been.

"There is a tide in the affairs of men which, taken at its flood, leads on to fortune." Let us not fail to take that tide as it comes and go on to the destiny that is, and should be, ours.

But you may say that destructive criticism is never helpful unless it is accompanied with constructive suggestions, and hence I feel it is my duty to point out to you in what way

I believe we can so shape our course as to be ready to meet the opportunity that is coming to us half-way.

First and foremost, then, since we must convince the outside world of the opportunity which we offer, let us modify our name, dear though it is to us all, so that it will make plain to everyone that we embrace in the field of our studies what is spoken of as "clinical medicine" as distinguished from laboratory work. Let us call our Association the American Climatological and Clinical Association, or, possibly better, the American Clinical and Climatological Association.

Next, let us amend our constitution so as to open our meetings to the discussion of all topics of general clinical medicine, especially the diseases of the lungs and heart, climatology and hydrology.

Thirdly, let us be careful in admitting new members to our Society, to remember Dr. Jacobi's warning and not to consider only, or chiefly, their good fellowship or the friendly recommendation of some gentleman, but let us consider more carefully than ever the work that they have done and are capable of doing, and their promise for the future, so as to be sure that they will add not merely to the social charm of our Association, of which we are so proud, but much more to its intellectual distinction, on which, after all, our reputation must be based, and, following the former practice of our sister society, the Laryngological, let us demand of all applicants for membership the submission of a thesis, to be approved before they can come up for election.

Finally, since a society cannot flourish unless the majority of its members take pains to attend meetings and show their interest by reading papers at intervals, let us more strictly enforce the rule that each member must be present and must read a paper before the Society at least every third year, thus assuring the active participation of all our members in the life of the organization.

And now, as I near the end of my remarks, I realize that some of my good friends who love this Association dearly, so that my criticism of it seems to them irreverent, will regard me as an iconoclast, and I feel that I must ask them to judge me kindly, realizing that my love for our Society is as deep and real as theirs, and that I am only anxious to see it continue the splendid career which has marked its past, and to avoid the errors that might endanger its future.

The tendencies which I have outlined, the remedies which I have suggested, I have been considering for four years or more, and I find that many others of our members have been thinking along the same lines. I have shown you that several of our Presidents have realized the need of some change, and I felt that the time was ripe to bring this matter to your attention. My earnest hope is that it may result in such action as will be to the benefit of the Society we love so much.

And now, in closing, let me express to you the very intense appreciation which I have felt at the honour you have done me in choosing me to stand on this platform to-day as your leader.

Possibly we have been too prone to dwell upon the wonderful solidarity and friendship which is peculiar to our Society; the members, feeling it so intensely, have spoken of it possibly too often, until outsiders, not comprehending it, have been inclined to scoff: but, great as is my pride at being for a while the Presiding Officer of such a body of men, proud as I am to receive that small portion of its reflected honour which falls on me for a while as your President, after all it is not merely or chiefly because this is a great and well-known Association, but first and foremost the knowledge that you, my friends, whom I have long known and cared for, have given me this evidence of your friendship and confidence, that most moves me to-day.

Intellectual opportunity must always remain the chief attraction of any medical association such as ours, but only when you have added to it the element of friendliness and kindly feeling that exists between us do you attain to the full possibilities of medical association.

Accept, then, my sincere thanks, and may this meeting, over which I am to have the honour to preside, be harmonious and profitable; may the papers laid before us keep up to the high level of the American Climatological Association; may our deliberations and decisions be wise and in the best interests of this Society, and may her name and her fame yearly increase.

ON THE AUSCULTATORY DETERMINATION OF EARLY PATHOLOGICAL CHANGES IN THE LUNGS.

BY HENRY SEWALL, M.D.
DENVER, COLORADO.

It is with the conscious risk of a charge of unwarranted temerity that I venture to invite you to re-enter a field of physical diagnosis in which each one has long delved as a master. My only excuse is that after twenty odd years of special personal study of the subject, of a sudden, the procedure of auscultatory examination became fraught with a coveted wealth of information concerning early recognition of intrathoracic disorders wholly due to a new point of view for the application of an old method.

The clinical results thus obtained suggest the experience of the farmer whose twenty bushels to the acre under the tillage of his ancestors has increased 100 per cent. with the application of a new line of thought to his labour. I am more encouraged to believe that my personal sense of enlightenment, derived through the procedure to be described, is founded on objective facts, because of experience with several patients who presented themselves shortly after having secured from real medical experts a report of perfect physical condition. That these patients were actually afflicted with pulmonary tuberculosis at the time mentioned was proved by recent positive sputa findings. The study

of the thoracic acoustics in these cases clearly bespoke pathological organic changes in the lungs, conditions which were amply verified on the skiagraphic plate.

Physical diagnosis demands different faculties and techniques according as the organic changes, whose existence it is its object to establish, are well advanced or only in their incipiency.

The adept who is able to announce the presence of a lobar pneumonia hours or even days before the development of the classic signs of the disease has cultivated art and imagination, as they are not expounded in the text-books.

The natural history of pulmonary tuberculosis, involving as it does gradual alteration by insensible steps of the normal structure, size and elasticity of the thoracic viscera, would seem to imply that we can never hope to determine by physical means the very advent of the disorder. If our physical examination reveals lesions which can be definitely ascribed to tuberculosis, the patient is already far advanced on a course which, if not interrupted, will lead to a fatal termination.

But there is a prologue to this pathologic history in which the plot of the story is foreshadowed by allusion and innuendo. By fortunate chance the alert critic of the prologue can, as at a rehearsal, completely change the *finale* of the contemplated play.

The earlier the stage at which we seek to determine morbid changes in the lungs the more important becomes the conscious appreciation of the physical signs which characterize the normal organs. This truism may be archaic, nevertheless I venture to assert that much of value lies still undetermined in the specific topographic distribution of the physiological and pathological physical signs of the chest. There is, for example, an assumed predilection of tuberculosis for the apices of the lungs. The lower half of the left

lung has long been to me a wonder area. Why, for example, do we so often find in tuberculosis a soggy, airless left base, which one is tempted to aspirate for a suspected effusion? Why, in tuberculosis, is the pulmonary tissue at the base and on the left border of the heart so apt to suddenly develop signs of consolidation which in the course of a few days abruptly clear up with a tympanitic note and evidence of cavity formation? It may be suspected that the topographic relations of the left auricle and of the pulmonary artery have much to do with the mechanics of the lower lobe of the left lung. X-ray studies of the chest show extraordinary variations in the prominence of those structures.

The work of Krœnig, Goldscheider, and others has of late years taught us that a peculiarly light percussion of the apices of the lungs is capable of disclosing the presence of a morbid pulmonary condition which could not be recognized by ordinary means.

It should, perhaps, be pointed out that a percussion dulness elicited in this way does not imply a deposit of such magnitude as to itself give the impression of a dull mass, but rather that the disease has altered the resonance of the lung, irrespective of the amount of deposit, so that its vibration response to percussion shall be characteristically altered. The advocates of this light percussion method specifically limit its usefulness to the apices of the lungs. The method, although of undeniable value, seems to me to unduly involve the personal equation of the examiner, and it demands for its employment mechanical skill of unusual degree. Moreover, the results of such percussion must depend in large measure on the amount and distribution of solid tissue outside the thorax on the two shoulders; bilateral differences in muscular rigidity may also, possibly, play a part. My own study has led me to the belief that through auscultation of the voice and whisper we have an unrivalled means for

the detection of minute tissue changes throughout the pulmonary region.

There is a close analogy between the conditions determining the acoustic properties of the chest and those on which depend the acoustic features of a music-hall. The details of structure in such a chamber vitally affecting its relations to sound have a minuteness that eludes calculation. We are safe in assuming that every change in the elasticity of the lungs and bronchial tubes, every deposit in contact with the vibrating air-columns, every abnormal congestion of the pulmonary parenchyma must alter the natural resonance of the lungs and be capable of detection by the ear.

These are the conditions which signalize pathological changes in the lungs apart from and preliminary to those adventitious signs known as râles. It is understood that adequate auscultation of the lungs involves a consideration of the findings from other methods of physical examination, especially the vocal fremitus and percussion resonance. Although the gradations in intensity, quality and distribution of the signs vary considerably among healthy subjects, nevertheless there is a fairly definite standard of the normal, departure from which is pretty sure to have a physical basis in disease.

It may be stated in brief that, as pointed out by Fetterolf* and others, the more marked tactile fremitus and vocal resonance distinguishing the right from the left apex of the normal lungs depends upon the immediate contiguity of the former structure with the resounding trachea, whose vibrations are thus transmitted transversely through the pulmonary tissue. Any infiltration of this tissue which improves its conducting or resonating properties must amplify and extend the sounds heard in auscultation.

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* *Arch. Int. Med.*, 1909, iii, p. 23.

Since it is very common, as shown on the X-ray plate, for the trachea to incline to the right of the middle line in its course through the chest, normal bronchophony at the right apex is variable in extent and intensity. It should, however, fade gradually from the vertebral column outward, posteriorly, and diminish rapidly below the spine of the scapula. The voice should not linger with an amphoric echo of heightened pitch, especially under pressure of the stethoscope. The whisper is, perhaps, normally confined to the inner third of the right apex laterally, and is limited by the inner lower border of the spine of the scapula below. At the left apex these acoustic characters are more limited in extent, and tend to be confined to the upper inner segment of the lung. In the front of the chest the same general statements as to sound distribution hold good; normal bronchophony and whisper are most distinct at about the inner halves of the first interspaces, fading thence outward and downward, more rapidly on the left. The normal distribution of vocal fremitus as determined by touch shows the vibrations to be more or less strong at the inner right apex, fading gradually in a lateral direction, and but little marked or absent at the left apex. At the pulmonary bases the fremitus when present is nearly equal on the two sides, possibly slightly more intense on the left. Fremitus is significant of vibration of the chest wall itself, and it gives rise to a powerful, resonant note under auscultation. This voicing of the chest wall proper is due to sympathetic vibrations, which reinforce especially the lower or "chest tones" of the larynx.

In 1890, assisted by Miss Pollard, I demonstrated that the fundamental tone of the chest wall could be altered to a certain extent by changing the respiratory phase and tension of the thorax.* In fact, the vocal artist actually

* *Journ. of Physiology*, 1890, xi, p. 159.

alters the vibration rate of the great thoracic resonator in a way to best reinforce the various notes of the musical scale. But as a whole, and under ordinary conditions, the fundamental tone of the chest wall is low in pitch, and the structure is thrown into sympathetic vibration most readily by the lower or "chest" tones of the register.

The importance of distinguishing these intrinsically mural from the purely visceral vibrations appealed to me many years ago, and I was able to show that the sympathetic vibrations of the chest wall itself could be essentially damped by the simple procedure of applying firm pressure to the bell of the stethoscope in contact with the skin.* For successful observations of this sort it is indispensable to use a purely air-conducting binaural stethoscope, the chest-piece of which shall be thick and heavy and not provided with a vibratile disk.

To fully appreciate the influence of this stethoscopic pressure and the part played by mural vibrations in vocalization one should, at the outset, auscultate both with and without pressure the base of a lung during the voicing of a "chest note." The voice heard under stethoscopic pressure loses much of its "resonance" and intensity and rises in pitch. As pointed out in another place,† the high "head notes" do not especially invoke mural vibrations, and hence are little altered by stethoscopic pressure. The mural vibrations being dampened, the ear perceives the laryngeal sounds only as transmitted and reinforced by the thoracic viscera. Any modification of the acoustic properties of the internal tissues may be detected by alteration of the normal sound. Two kinds of sound are made use of in auscultation, namely, the voice and the whisper. The former is essentially a musical note due to periodic vibrations of the vocal cords,

* *New York Med. Journ.*, 1897, lxvi.

† *Amer. Journ. Med. Sci.*, 1913, cxlv, p. 234.

and is prone to set up sympathetic vibrations in all elastic bodies impressed by it. The latter is "noise" or sound produced by non-periodic vibrations of several structures, including, probably, the false cords and the soft palate. Non-periodic vibrations are ill-calculated to induce sympathetic resonance. Accordingly, we should not expect to find whispered sound reinforced by sympathetic tissue vibration; it is heard only as *conducted* from the site of its production. Thus, a superficial excavation in the lung may be accurately outlined through the whispered sound, but the "cavernous voice" tends to spread beyond the limits of the cavity.

On auscultating over the *manubrium sterni* whispered sound is heard with greater or less intensity. When such sound persists under firm pressure with the stethoscope, I am convinced that we have demonstrated an abnormal mass of conducting tissue, vascular or glandular, conveying the vibrations of the underlying trachea directly to the surface. It was an important observation of Manges that a distinguishing mark of whispered sound is its lack of transmission.* This is a way of expressing the fact that whisper is incapable of exciting vibration in the chest-wall when the normal lung is interposed between the internal site of vibration and the surface. Normal lung tissue is an excellent "silencer" of sound. Air channels penetrating stiff, sclerosed tissue are admirably adapted to transmit the vibrations of whisper. The morbid changes which are prone to involve the roots of the lungs even in early pulmonary tuberculosis—the fibrous infiltration and glandular enlargement in immediate contiguity with the back wall of the chest at about the level of the scapular spines—offer a rational explanation of the characteristic transmission of the whisper in this area.

A. d'Espine has long contended that bronchial adeno-

* MANGES: "Archives of Diagnosis," 1912, v, p. 208.

pathy in the child can be readily detected through auscultation.* "The special sign which permits us to diagnose the condition at an early period is based on the auscultation of the changes in the voice sounds at the level of the seventh cervical or first dorsal vertebræ. . . . The sign is most obvious when one makes the child speak or count in a low voice. The voice is then accompanied by an added whispering sound localized to one or two vertebræ, or possibly extending to the fourth or fifth."

B. Zabel† endorses this method as superior to all others for the early recognition of bronchial adenopathy, and points out, as did d'Espine, that when the diagnosis is uncertain by means of the voice it may be established through use of the whisper.

It is common in acute pulmonary affections, as of lobar pneumonia or influenza, to find sharply circumscribed areas of transmitted whisper in the lower parts of the chest. Such an auscultatory condition may occur with none of the percussion dulness denoting consolidation. I can think of no reasonable explanation of the fact, except it signifies hyperæmia of the lung tissue through which its acoustic properties have been modified. The X-ray plates of such chests made for me by Dr. S. B. Childs have all presented in the area in question a uniform haze such as might be produced by a thin sheet of effusion, though there could be no suspicion of such an occurrence. Hyperæmia of the pulmonary capillaries seems to be the most reasonable explanation of the finding.

A careful study of certain labile cases of chronic pulmonary tuberculosis will disclose now and then, especially after undue physical exertion, or at the outset of a "cold,"

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* A. D'ESPINE: "Early Diagnosis of Bronchial Adenopathy in the Child," *Brit. Med. Journ.*, 1910, iii, p. 1136.

† B. ZABEL: *Münch. med. Woch.*, 1912, lix, p. 2664.

new areas of whisper transmission over one or the other lower lobes of the lungs. If the patient is kept at rest the whisper transmission in these areas gradually fails, and coincidently there occurs a clinical improvement in the patient's condition. Indeed, I have been led to combine all these facts in a working generalization, namely, that the earliest morbid signs of pulmonary infection are due to more or less localized hyperæmias, which, if sufficiently intense and near the surface, may be recognized by transmission of whispered sound, but in any case, as will be noted below, may be manifested by an alteration in the normal vocal resonance. The prognostic importance of such hyperæmia itself is probably wholly good, for it is presumably a salutary reaction against toxic invasion, and the therapeutic indication is mandatory; it is absolute physical and mental rest. The relatively acute pathological condition referred to above is, of course, not to be confused with that more chronic state having nearly identical auscultatory signs in which there is evidence of pneumonic consolidation or fibroid infiltration.

Turning now to the auscultation of the voice, we find, as should be expected on theoretical grounds, that it is marvellously capable of registering modifications in the acoustic properties of the thoracic viscera. When mural vibrations are damped by stethoscopic pressure we are in a position to apprehend the changes in the internal organs. In general, it may be said that with intense congestion of the lungs or such tissue changes as occur in early pulmonary tuberculosis, the voice takes on a more or less amphoric or tracheal character, and it tends to become more distinct, prolonged, raised in pitch, and nearer the ear with pressure upon the stethoscope. When the patient counts "one, two, three" there is a tendency for the voice to linger with a bleating echo, which is exaggerated by stethoscopic pressure. Judgment and practice on the part of the examiner are essential to

enable him to discriminate between the widely diffused ringing bronchophony transmitted by air tubes traversing a somewhat hyperæmic but normal lung—as shown by the skiagraph—and those similar but differently distributed signs of incipient pulmonary pathologic change. Deductions from the results of so delicate a method as that under discussion, must needs be guided by careful reasoning for the right interpretation of the facts it discloses. Nevertheless, as pointed out before, anyone fit to carry on a physical examination can, with a little application, familiarize himself with the normal voice sounds and their distribution with and without pressure of an appropriate form of stethoscope, and thus be in a position to detect changes in the pulmonary and bronchial apparatus which elude all other methods of examination.

X-ray pictures of the lungs may prove of the highest value, either in reassuring one as to the absence of suspected trouble or in giving warning of a low ratio of vital resistance to infection virulence. For example, a patient who was thought to have probably recovered from a bilateral pulmonary tuberculosis came for examination after an absence of some months. There was now so marked an exaggeration, with amphoric tone, of voice and whisper in the upper chest that the possibility of rapid excavation had to be considered. An X-ray plate, however, gave complete reassurance, for it showed the lungs in good condition, but plentifully marked with evidences of sclerotic healing—a tissue change which tends to exaggerate visceral vibrations and render the sound amphoric.

I would like once more to specifically voice the opinion that skiagraphic examination of the chest through the photographic negative has already become an indispensable adjunct to the more familiar procedures of physical diagnosis.

Prognosis and therapeusis in early stages of tuberculosis

find, in my experience, extraordinary illumination under the X-ray. In "good" cases the skiagraphic evidences of scar tissue, opaque scattered flakes and strands, and glandular calcification are apt to be profuse even in the absence of ordinary morbid, physical and clinical signs. In "bad" cases there may be softly mottled or "mossy" areas indicating infiltration without sharp shadows of fibroid tissue or glandular calcification. Clinical experience with such subjects has shown me that they are exceedingly liable to retrograde with the slightest hygienic dereliction; the *rest* cure seems specifically necessary for them.

Briefly summing up the evidences that have been presented, we may conclude: (1) That auscultation is capable of giving the earliest objective information of physical changes in the lungs; (2) that such study demands analysis of the sound into those vibrations due to resonance of the viscera and those of the chest wall, and that the latter vibrations can be damped by pressure of an appropriate form of stethoscope; (3) that vibrations transmitting the whisper are confined almost wholly to the viscera; (4) that the modification of the voice sounds, by which their quality becomes more amphoric and their duration prolonged into an echo, are the striking characters which, when accentuated by stethoscopic pressure, indicate pathological changes in the viscera; (5) that the character and distribution of vocal signs over the normal chest are sufficiently constant, so that a topographic study of the chest by auscultation may definitely suggest, through recognition of departures from the normal, the intensity and distribution of morbid changes within the lungs, even when these changes are too slight to appeal to the senses through any other method.

DISCUSSION.

Dr. J. N. Hall (Denver, Colo.): For nearly twenty years Dr. Sewall and I have had opposite sides of the same ward of the City Hospital, and I have seen the cases as they came in. I wish to testify to the striking work that he has done in the ward. I have seen him diagnose cases which were positively unique.

Dr. W. H. Swan (Colorado Springs, Colo.): Dr. Sewall speaks of the value of the X-ray in showing the early stages of tuberculosis. I think we sometimes overlook very marked signs by ordinary examination when the X-ray shows them very distinctly. I recall now a case in which several members of this Society had failed to recognize a well-marked and large cavity, but the X-ray showed a very distinct cavity, with definite outline and thickened wall. Even after the cavity was demonstrated by the X-ray there were absolutely no physical signs to show it. This must lead us to be very cautious indeed as to many of our physical findings.

Dr. J. Madison Taylor (Philadelphia, Pa.): Dr. Sewall's lucid presentation brings forcefully to my mind the extraordinary deficiencies many medical students (not to mention some practitioners) exhibit in fundamental physiodynamics. How can they appreciate and apply these principles, unless originally grounded in them? It is my conviction that this subject should receive specific attention in all medical schools; if not as a separate study, then as part of the training in physiology. In the preliminary studies, whatever else is required, I would urge the imperative need of physics as applied to biology and physiology.

Dr. L. B. Wilson (Rochester, Minn.): I would like to bear witness to the great value of the whisper voice. I find that it is much easier to map out by the whisper than by any other ordinary means of physical diagnosis.

Dr. William LeRoy Dunn (Asheville, N.C.): I would like to know what particular formula the doctor has found especially adapted in this whisper voice in bringing out the best evidence.

Dr. Sewall: I direct the patient to repeat the words, "one, two, three," and I note the prolongation of the sound from one syllable to the other, as well as its change in quality. Mr. Chairman, my friends have been very kind in their comments upon my paper. I would have been rather better pleased had more antagonism been developed. If what I have said is true, there is rarely need for any tuberculosis to reach the open stage. Just think of it; there is no need for tuberculosis ever, except in certain particular instances, to reach the open stage. My thesis is that the procedures of physical diagnosis within our reach are capable in the vast majority of cases of disclosing the presence of pulmonary and glandular tuberculosis in a stage so early that not only is the patient still non-infective, but is capable of readily responding to the curative measures of hygienic living.

THE ANATOMIC CAUSES FOR THE DIFFERENCES IN THE PHYSICAL SIGNS OVER THE UPPER LOBES OF THE LUNGS.

By GEORGE WILLIAM NORRIS, A.B., M.D.

PHILADELPHIA.

In different text-books on physical diagnosis very various and, for the most part, unsatisfactory reasons are given for the difference in the physical signs obtained when the upper portions of the lungs on the two sides of the chest are compared by palpation, percussion and auscultation. The following illustrations made from frozen sections prepared by Dr. George Fetterolf depict some of the reasons which we consider accountable for the above-mentioned differences.

I.—Vocal Fremitus and Resonance.

As was pointed out by Fetterolf,* vocal fremitus is more intense on the right side because the trachea is placed in intimate contact with the apex of the lung. On the left side these structures are separated to the extent of $\frac{3}{4}$ in. to $1\frac{1}{2}$ in. by the interposition of the aorta, the left common carotid, and subclavian arteries, together with a variable amount of lymphatic and alveolar tissue (see fig. 1). Some thirty or more bodies have now been sectioned for different purposes,

* Fetterolf, G.: "The Anatomic Explanation of the Greater Amount of Vocal Fremitus and Vocal Resonance Normally Found at the Right Apex of the Lung," *Arch. Int. Med.*, February, 1909.

and this relation of the trachea to the right apex has been constantly found.

II.—The Percussion Note.

Over the right upper lung the percussion note is often normally less resonant, higher pitched, and slightly vesiculo-tympanitic in character. This is due to three factors.*

First.—As regards the anterior position of the vessels of

Fig. 1.—Mediastinal surface of lungs hardened before removal. This photograph shows (*a*) the groove produced in the right apex by the trachea; (*b*) the relatively anterior position of the vessels on the right side; and (*c*) the smaller size of the right apex. *AG*, azygos groove; *VG*, grooves for superior vena cava, innominate vein, and subclavian vessels; *TG*, tracheal groove; *SG*, subclavian groove; *AAG*, aortic groove; *CI*, cardiac impression.

the right side. The first branch, better called the anterior branch, of the horizontal part of the aortic arch is the innominate artery, destined for the supply of the right head

* Fetterolf, G., and Norris, G. W.: "The Anatomic Explanation of the Relatively Less Resonant, Higher Pitched, Vesiculo-tympanitic Percussion Note Normally Found at the Right Pulmonary Apex," *Amer. Journ. Med. Sci.*, cxliii, 1912, p. 637.

FIG. 2.—Anterior view of lungs hardened before removal. This photograph shows the anterior position of the groove for the subclavian vessels on the right side, compared with the more superior position on the left. *SG*, subclavian groove.

FIG. 3.—Lateral view of lungs hardened before removal. This photograph shows the deeper vascular groove and the smaller size of the right apex as compared with the left.

and the right upper extremity. Arising, as it does, to the left of the median line, it is compelled to pass anterior to the trachea in order to reach its destination. This position of its origin is responsible for the anterior situation of the vessels in relation to the right apex. Having crossed the

FIG. 4.—Frontal section of the thorax in the axis of the trachea. This photograph shows (*a*) the contact of the right upper lobe with the trachea; (*b*) the mesial position of the left subclavian artery in relation to the left upper lobe; and (*c*) the essentially anterior position of the right subclavian in relation to the right upper lobe. *RUL*, right upper lobe; *RSA*, right subclavian artery; *RCA*, right common carotid artery; *T*, trachea; *E*, œsophagus; *LCA*, left common carotid artery; *LSA*, left subclavian artery; *LMA*, left internal mammary artery; *A*, aorta; *LUL*, left upper lobe; *LPV*, left inferior pulmonary vein; *CT*, carina tracheæ; *RVN*, right vagus nerve. (Reproduced from the *Archives of Internal Medicine*, February, 1909.)

anterior aspect of the trachea and turned somewhat posteriorly, it divides into its terminal branches, the right common carotid and the right subclavian, the former of

which does not concern us. The latter passes obliquely upward and *backward* to reach its groove on the upper surface of the first rib. In its course it produces a deep sulcus in the anterior aspect of the pulmonary apex. (See figs. 5 and 6.)

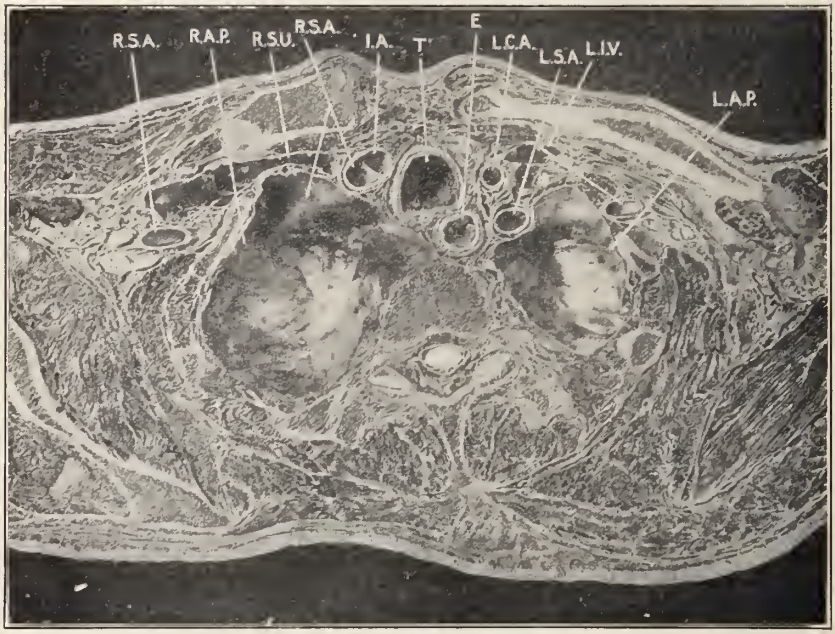

FIG. 5.—Section through the upper part of the thorax, viewed from below. The line of section is not exactly horizontal, a slightly lower plane being reached on the right side than on the left. In order to show the apical parietal pleura, the pulmonary apices have been removed. There can be noted in this specimen the beginning contact of the right pleura with the trachea, and the anterior position of the innominate artery, whose bifurcation is well shown. On the left side, the wide separation of the pleura from the trachea by means of the large arteries, œsophagus, and areolar tissue can readily be seen. There can be seen well the deep position of origin and the obliquely anterior course of the left subclavian artery. *T*, trachea; *E*, œsophagus; *RAP*, right apical pleura; *LAP*, left apical pleura; *IA*, innominate artery, dividing into *RSA*, right subclavian artery, and *RCA*, right common carotid artery; *LSA*, left subclavian artery; *LCA*, left common carotid artery; *RSV*, right subclavian vein; *LIV*, left innominate vein.

The second branch of the horizontal part of the arch is the left common carotid. It passes upward at the side of the

trachea, between the trachea and the left apex, and enters the neck.

The third, or posterior, branch of the horizontal part of the arch is the left subclavian. This vessel is given off well back in the mediastinum, opposite the posterior surface of the trachea, or even at the left side of the œsophagus. It

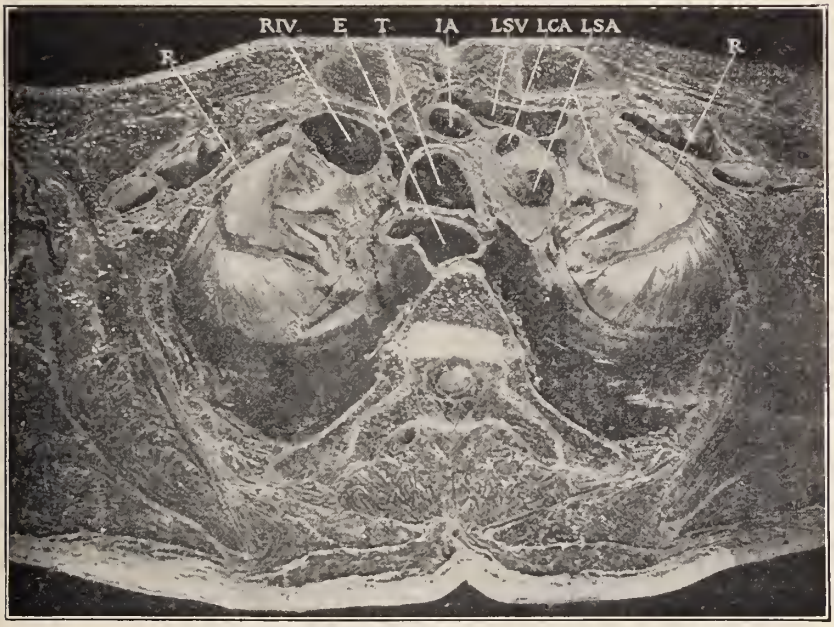

FIG. 6.—Horizontal section of the thorax just above the sternoclavicular articulation. The pleura has been dissected away. This photograph shows (*a*) the anterior position of the innominate artery; (*b*) the posterior position of the left subclavian artery; and (*c*) the anterior position of the right innominate vein. *R*, first rib; *RIV*, right innominate vein; *E*, œsophagus; *T*, trachea; *IA*, innominate artery; *LSV*, left subclavian vein; *LCA*, left common carotid artery; *LSA*, left subclavian artery.

arises so deeply that it is compelled to pass slightly *forward* as well as laterally to reach its groove on the first rib. In the greater part of its thoracic course it occupies the layer of tissue between the left upper lobe and the trachea. Nearing

the first rib, it produces in the left apex a groove which is shallower and nearer the summit than on the right side.

The same principle applies to the veins. The right apex is encroached upon and markedly grooved by the subclavian and innominate veins, these grooves being continuous with one another, and situated in front of the groove for the subclavian artery. On the left side the subclavian vein produces a sulcus in front of that of the artery, but being more mesial it does not encroach to so great an extent on the apex.

Second.—The smaller size of the right apex as compared to the left. This is dependent on the facts enumerated in the previous section. On the right side the vessels pass in front, and occupy space which on the left side is occupied by lung tissue.

The shape and size of the pulmonary apices are modified by the vessels in the same way as would be two small hills over which roads were cut. In one case, as on the right side, the road is cut over the side of the hill and requires excavation. On the other side, corresponding to the left apex, the road is carried more nearly over the summit, and comparatively no excavation is needed. The result, as far as the apices are concerned, is to cause the former to be smaller than the latter, and to give the right apex a more conical, and the left a more dome-like shape. (See figs. 1, 2 and 3.)

Third.—The apposition to the inner surface of the right apex of the trachea and to the left of solid tissue. This has been emphasized by one of us in a previous paper, and is demonstrated clearly in figs. 4 and 5.

III.—Auscultation.

The breath sounds at the right apex are normally more harsh and higher-pitched than on the left. They contain more of the bronchial element. Slight degrees of whispered

pectoriloquy are more common in normal lungs than on the left.

These differences are again due to the close proximity of the trachea to the apex on the right side, which adds a harshness, a bronchial element, and a higher-pitched tone to the soft, low-pitched blowing quality of the vesicular murmur.*

DISCUSSION.

Dr. WILLIAM C. WHITE (Pittsburg, Pa.): I have seen the work of Dr. Norris and Dr. Landis, and I think it is one of the most distinct advances in teaching medicine and diagnosis that has been made for a number of years. I would offer a suggestion, without a note of criticism in it, that the question of percussion of the posterior of the chest is not a question of comparison between the back and the front, but a question of percussion between the chest in a diseased condition, and the normal chest. It would be just as possible to acquire adeptness in comparison between a diseased condition and normal condition even though there be a large muscle and bone on the back wall.

* The illustrations were chosen from those shown as lantern slides at the meeting.

THE EARLY LESIONS OF TUBERCULOSIS IN THE LUNGS.

By PHILIP KING BROWN, M.D.
SAN FRANCISCO.

We have all seen early cases of tuberculosis in which the clinical signs were difficult to make out, and yet the patients go on rapidly to frank lesions with cavity formation, in spite of all treatment. There may have been no recent exposure, but a death in the family years before suggests the fact that tubercle bacilli found lodgment somewhere at that time, reposing latently for years, only to make trouble at a later date. Then there is the hæmorrhage which may bring to the doctor for examination a patient with evidence of a very limited infection in the apex, which, under care, shows no definite clinical signs in a few months, and even at the time of the hæmorrhage it may not be possible to locate the lesion. With no signs but ease of fatigue, slight loss of weight and subnormal temperature, we find case after case with no cough or expectoration, but reacting to tuberculin. All these conditions and more have been presented to us time and again, and we follow enough of each group to make us guarded in prognosis, even in cases of seeming mild infection.

Are we to consider these early cases with their differing progress expressions of varying resistance of patents, or varying virulence of the infecting bacilli, or is there some

more important factor still? It is to call attention to the frequency of primary involvement of the glands at the root of the lungs, the tracheo-bronchial groups, and the extension of the disease along the lymph channels in the reverse direction of the current that this subject is presented. With no thought of denying direct air-borne infection of lung tissue proper, rupture of softened nodules, glandular or otherwise, into the blood-stream or bronchi, there are still the vast majority of cases of lung tuberculosis which, if seen early and subject to exhaustive clinical and X-ray examination, do not apparently belong to the types of infection which result rapidly in intense but localized lesions. In a series of some 200 early cases in young women seen in the last year and a half in connection with examinations for entrance to Arequipa Sanatorium for early cases in working girls, and in following 100 of them for an average period of four months, certain facts were definitely established. (In all the cases instantaneous X-ray plates were used to check up the physical examinations and tuberculin tests.)

(1) Enlargement of the tracheo-bronchial glands was a regular occurrence when the subcutaneous tuberculin test was positive.

(2) Every early case showing tubercle bacilli in the sputum showed not only the glandular enlargement but thickening of the bronchial tree extending outward from the hilus, frequently terminating in a definitely involved area.

(3) Cases not positively diagnosed by physical signs and without bacilli in the sputum—but with cough, loss of weight, menstrual and gastric disorders, frequently showed striking changes in the X-ray plates, the bronchial tree being marked by a group of glands at the hilus, and the shadow from the group being thickened and studded with nodules along its course. The nodules generally were smaller as the periphery of the lung was approached.

(4) When there were evidences clinically and in the plates of an active process near the periphery, the linear markings did not confine themselves to the exact area of activity, but a segment frequently much wider showed the denser shadows converging to the hilus glands. Had the peripheral lesions been primary, it is hard to see why the thickenings and nodules were not confined to the area drained by the lymphatics.

The inference is drawn from these facts that the extension of the infection is centrifugal and not centripetal.

Further proof of this was found in the testing of thirty-one children of known tuberculous parents, but without physical signs of tuberculosis. In fifteen the skin tests were positive, and in sixteen negative. Every one of the fifteen positive cases had very definite enlargement of the hilus glands, and in none of the sixteen negative cases were they marked, although they were noticeable in a few of the children. Because glands are enlarged they are by no means necessarily tuberculous, but the results of the comparison of skin reaction and X-ray plates in these two groups of children were very suggestive

A review of 3,000 autopsy records of the Pathological Department of the Massachusetts General Hospital, made by Drs. J. H. Wright and Oscar Richardson, showed that where note was made of the condition of the mediastinal and bronchial glands in obsolete cases of tuberculosis, they were found involved alone four times more frequently than were the lungs (600 to 156). There is no question that even in adult life the condition of the glands is of far more importance than has been considered by clinicians.

The records of autopsies on children show this still more strikingly, and tuberculosis of the bronchial glands is absolutely the commonest seat of infection with the Koch bacillus. Ghon, almost alone of recent observers, regards the

infection of the lung tissue as primary and the gland involvement as secondary. Against this view are arrayed Escherich, Shennan, Comby, Harbitz and numerous others, all with records of two to four times as many autopsies. But no mention is made by any of them of the manner in which the gland infection results in a spread to the lung further than to refer to the caseous tubercular broncho-pneumonias of childhood resulting from rupture of tuberculous glands into bronchi.

Still more light is thrown on the question by an examination of the records of slaughter-houses under federal inspection. Here one finds again that the tracheal and bronchial glands in cattle, the same groups in hogs, plus the tonsils and cervicals, comprise the largest source of primary infection, and are involved four to eight times more frequently than the lungs.

Must we not consider that from the clinical point of view the vast majority of cases of lung tuberculosis in human beings begin in a very limited area of the lung, and dealt with early are favourably influenced? Not so the tuberculosis which follows rupture of a softened tubercular gland into a blood-vessel or large bronchus, nor can we find in the idea of direct air-borne infection much that explains the clinical or pathological findings in a majority of cases. We have been told that the right lung is more frequently involved than the left because, on account of the angle made by the right bronchus, air enters it more easily. In the next breath we get the suggestion that the apex is the seat of predilection for the disease, because air enters there less easily than elsewhere in the lung. Surely this explanation is not satisfying. As a matter of fact, air-borne infection results in the involvement of the glands, for we have been shown repeatedly that tubercle bacilli pass the intact mucous membrane of the air tract as well as the digestive tract.

It is not necessary to throw aside the idea of direct infection of the lung from air, but it certainly does not occur as often as we are led to suppose. On the other hand, there seems excellent physical reason for disturbed lymphatic as well as arterial circulation in the lung apex, and a retrograde infection along the lymphatics from the tracheo-bronchial glands may be the result.

DISCUSSION.

Dr. SEWALL: I was very much interested in the exhibition that Dr. Brown has made. The doctor did not mention the work of a man not known to me personally, Dr. Ghon, of Vienna, who has recently published a book on the earliest locus of tuberculosis in a child. He comes to the conclusion that the deposit of tuberculous material in the child is frequently not in the gland at all, but in the lower lobe of the lung. The X-ray picture has in my rather limited experience shown to me that when you can detect a lesion in the apex by X-ray you almost invariably find in the lower lobe of the lung a modification of the circulation, I mean of those shadows which form a part of the bronchial tree, modifications of the circulation which lead one to suspect tuberculous deposit.

Dr. GEORGE W. NORRIS: In the presence of so many gentlemen who limit their practice to tuberculosis, and are therefore in a better position to speak, I rise with hesitancy to express my doubt as to the value of the X-ray in the diagnosis in earlier cases of tuberculosis. It has been in my experience that I have turned over a number of early cases to experts, and have in a discouragingly large number received the reply that the lesion is on the opposite side from that on which I diagnosed it. Now, of course, I may have been mistaken, but I do not think I was mistaken all the time, and I must say that so far as my personal experience goes I do not regard the X-ray as of any particular diagnostic value in the early stages of tuberculosis. I think that when the disease is advanced it helps to a certain extent. There are many cases in which we can make no diagnosis when we have simply glandular involvement. The X-ray picture shows the glands enlarged; we look in another case in which there are no symptoms of tuberculosis, and also find the glands enlarged. I do not think that it is possible by the X-ray to say that these glands are tuberculous. Another point is to me curious. The idea that the Röntgenographers have with regard to the congestion of the lung. They send back the report that the right lower lobe is congested when there are absolutely no indications. Now I cannot conceive of a right lung being sufficiently congested to be demonstrable by means of the X-ray without producing some physical sign.

Dr. PHILIP KING BROWN: Dr. Jordan, who is the radiographer of Guy's Hospital, reported 150 consecutive cases of X-ray examination recently in which the bacterial findings were positive, and he reports that in the X-ray findings the apices seemed to be involved alone thirty-two times, the roots alone fifty-nine times, and the apices and bronchial glands fifty-nine times. So that I think that that alone is evidence of the fact that a great deal of tuberculosis is so deep-seated that you cannot easily discover it by physical examination. I have examined cases in which I could not tell where the lesion was. I believe that every one of you could recount just such experiences. If it be true, as Dr. Jordan says, in the findings by X-ray that the glands alone showed the signs in fifty-nine cases I do not see how Dr. Norris can make the statement that he does; and if it be further true that practically every child that dies in the Babies' Hospital in New York has bronchial gland tuberculosis, and Osler in his text-book calls attention to 127 such consecutive cases, with very little tuberculosis elsewhere, the proportion who have it elsewhere being very small, it seems to me also true that physical examination has a good many limitations.

BLOOD-SPITTING IN HEART DISEASE.

By J. C. WILSON, M.D.

PHILADELPHIA.

Blood-spitting occurs in association with heart disease under various conditions. Clinically, three principal groups of cases may be differentiated :—

(1) Those in which, with an antecedent or concomitant lesion of the heart, the hæmoptysis is essentially of pulmonary origin, as in tuberculosis, croupous, and sometimes bronchopneumonia, infarct, acute and chronic bronchitis, emphysema, bronchiectasis, and malignant growths.

(2) Those in which vascular structures are the seat of hæmorrhage, as aneurism, erosions caused by foreign bodies or malignant growths, and primary and secondary diseases of the blood.

(3) Those which are primarily and essentially of cardiac origin.

This communication concerns the last of these. It does not fall within its scope to enumerate, much less discuss, other conditions which are occasionally attended by hæmoptysis.

The condition under consideration is best studied in office work and in private practice. For this there are several reasons. The cases are mostly ambulatory, since the compensation is fairly well maintained and the hæmoptysis very often slight, and shows itself only at considerable intervals of time. When compensation fails and the ultimate dyscrasia

occurs in heart cases, bleeding is rare; when the hæmorrhage is abundant, as is by no means unusual, the attack is apt to be of short duration. A few hours of rest in bed brings it to an end, and in a day or two the patient is up and about feeling none the worse, often distinctly the better for the blood loss. Such cases, when received into the hospital ward, are soon discharged and promptly forgotten. The occasional scanty blood-spitting, which is much more common, does not usually interest the overworked dispensary physician, who too often misses its significance, much as it alarms and distresses the patient. Many of the cases are referred to the consultant for an opinion as to the diagnosis, the recurring hæmoptysis causing much alarm, while on the one hand the clinical phenomena relating to the lungs are obscure and the laboratory findings negative, and the cardiac signs and symptoms are not appreciated. In fact the significance of blood-spitting as a manifestation of heart disease is too often wholly overlooked in general practice. To the internist who has made it the subject of special study it is of the highest importance.

This symptomatic manifestation of valvular disease, especially of mitral stenosis, is much more common than the text-books would indicate. In fact most of them, and some of the special treatises on diseases of the heart, do not mention it at all. Among the more recent authoritative statements in regard to the matter are those in the article on mitral obstruction by the late A. E. Sansom,* and in James Mackenzie's remarkable monograph on diseases of the heart.† The former writer states, after calling attention to the fact that epistaxis is one of the earliest symptoms to attract attention in this form of chronic valvular disease,

* Revised by G. A. Gibson in the later edition of Allbutt and Rolleston's "System of Medicine," 1909, vi. p. 366.

† MACKENZIE, JAMES: "Diseases of the Heart," 1908, p. 226.

that hæmoptysis has often been recorded, and that it also occurs in the course of the lung affections in this disease; while the latter affirms that "at various stages patients may be seized with great bleeding from the lungs. Here doubtless the cause is the back-pressure in the pulmonary circulation and rupture of the blood-vessels." And he adds, "As a rule this is a grave sign, the patient dying sometimes shortly after an attack." Hæmoptysis in mitral stenosis, though often very alarming, is usually followed by abatement of the cardiac symptoms, and sometimes by prolonged improvement. I have recently reported two cases which are interesting in this connection.*

A woman, aged 52, the mother of four children, consulted me in 1906 for blood-spitting in small amounts, from which she had suffered for ten years. There were well-characterized signs of mitral stenosis. She had never had rheumatism, chorea, or puerperal sepsis. There had been no habitual cough, and there were neither symptoms nor signs of pulmonary tuberculosis. Tubercle bacilli were not found in the sputum. Her weight had risen from 136 to 150 lb. She came again on September 22, 1910, stating that she had been entirely free from blood-spitting until May 20, 1910, when she had a single copious bleeding amounting to "at least a quart." Since then she had been well. There were at this recent examination no pulmonary signs whatever, and she had neither cough nor expectoration. The signs of mitral disease were the same as four years previously, and she complained of slight dyspnœa on exertion, but nothing else.

A Jewess, aged 45, seen in consultation, was unable to recall any attack of rheumatism, and regarded herself as well except for some shortness of breath on exertion, which she attributed to increasing weight. Coming home from the theatre this patient was seized with profuse hæmoptysis,

* Wilson, J. C.: *Penn. Med. Journ.*, May, 1911, p. 596.

which continued until the following day. There were no signs or symptoms of pulmonary tuberculosis, and no tubercle bacilli were found in the clots, but a coarse presystolic thrill, a loud rumbling murmur at the same period in the cardiac cycle, and absolute absence of cough or expectoration after the hæmorrhage ceased, which it presently did, showed that it had been due to stenosis of the mitral valve. After several months this patient remained free from hæmoptysis.

I have reported a remarkable and probably unique case of contusion of the chest followed by the development of presystolic murmur and thrill and recurrent copious hæmoptysis, which terminated fatally from continuous hæmorrhage nineteen months after the injury.*

These hæmorrhagic cases of mitral stenosis are very often erroneously regarded as irregular or obscure cases of pulmonary tuberculosis.

Blood-spitting, especially when the amount has been small and the intervals of its occurrence have been prolonged, is often overlooked in the history-taking of heart cases. This is due to the fact that for the very reasons stated, though very alarming to the patient at first, through familiarity it soon becomes less important to him, especially when dyspnœa, precordial distress and œdema develop as compensation fails.

Jex-Blake,† in a recent study of 2,400 cases seen in the out-patient service of the Brompton Hospital for Consumption and Diseases of the Chest, in two years found that at least 909 had suffered from hæmoptysis at one time or another during the course of the disease. Of this number, in 497 instances the hæmorrhage was ascribed to pulmonary tuberculosis, in 333 to other affections of the lungs and bronchi, and in 30 to lesions of the heart. In the remaining 59 the blood-spitting was attributed to various conditions, but it is

* *Journ. Amer. Med. Assoc.*, February 10, 1912, lviii, pp. 405 and 406.

† *Practitioner*, November, 1911, pp. 616 *et seq.*

interesting to note that in 35 cases the cause was set down under the heading "Tonsils and Adenoids," since chronic hyperæmia of the structures of the faucial inlet is of occasional occurrence in valvular disease of the heart. Among the 2,400 patients on whom these statistics are based 31 had mitral disease, and 25 of these hæmoptysis. Eight presented the signs of mitral insufficiency, and all of these had had blood-spitting; 11 the signs of mitral stenosis, and of these 7 had had blood-spitting; while 12 had insufficiency and stenosis combined, and 10 had suffered from blood-spitting. There were two cases of congenital pulmonary stenosis, in one of which blood-spitting had occurred. In 5 of the 25 cases of mitral disease there was associated pulmonary tuberculosis; in two bronchitis and emphysema, and in one fibroid lung—conditions in which hæmoptysis is common in the absence of heart lesions.

There were five cases of aortic insufficiency, in four of which hæmorrhage had occurred, and of these one had an associated pulmonary tuberculosis, and one bronchitis and emphysema.

Analyses of a series of cases, not including pneumonia, coming under my observation in office and consultation practice during a period recently terminated give the following results:—

Whole number of cases			304
Cases in which hæmoptysis was noted			37
Tuberculosis	5		
Bronchitis and emphysema	4		
Tuberculosis and mitral stenosis	1	—	10
Cases of heart disease			47
Heart disease with blood-spitting			27
Mitral insufficiency	23		
,, ,, with blood-spitting	10		
,, stenosis	21		
,, ,, with blood-spitting	14		
Aortic stenosis	3		
,, ,, with blood-spitting	2		
Congenital heart disease with blood-spitting	1		

A very large proportion of mitral lesions cause at the same time insufficiency and stenosis, with to-and-fro murmurs. The above classification is based upon the relative prominence of the respective signs. There was no case of aortic insufficiency in this series.

The clinical variations range from a mere trace of blood —blood-streaked sputa usually occurring in the course of a bronchial catarrh of moderate intensity—to copious hæmoptysis, which may terminate in death. The fatal issue is, however, uncommon.

When the tendency to blood-splitting has manifested itself in valvular disease of the heart, hæmorrhage is liable to occur to some degree in any acute or chronic affection of the lungs.

In fact, although cardiac disease is frequently the primary cause of blood-spitting it is never the direct cause, but always acts through the rupture of over-distended capillaries or venules or the occurrence of embolism or infarction. It is evident, therefore, that any lesion of the bronchi or lung parenchyma may cause bleeding. The difficulties in diagnosis have already been alluded to. They are more apparent than real, and may be minimized by a systematic method.

No examination of the lungs is complete in the absence of an examination of the heart, and the converse of this proposition is equally true.

The errors commonly arise from the assumption that blood-spitting is a manifestation of primary disease of the lungs, and ignorance of the fact that it is often a sign of obstruction or insufficiency of the valve mechanism of the heart, resulting in the transference of blood-pressure from the arterial to the venous side of the circulation.

In fact, the whole progression of ills from the trifling and transient dyspnœa upon unusual effort, which is the first

sign of cardiac inadequacy, to the terminal dyscrasia or arrest of the heart in asystole constitutes a series of symptoms of lowered arterial and heightened venous pressure. Whether the effects of this derangement of balance fall upon one set of organs or another or uniformly upon all depends upon anatomical peculiarities of which we have a very incomplete knowledge. When they are chiefly manifested upon the vascular structures of the lungs, blood-spitting is a more or less conspicuous symptom.

The diagnosis must be made by exclusion.

The immediate prognosis as to the hæmoptysis is usually favourable. The fatal cases are rare. The remote prognosis is that of the cardiac lesion. A medical student had a very profuse hæmoptysis. No signs of phthisis, but well-marked evidences of mitral stenosis. In a short time he was able to continue his studies, was graduated, and entered upon practice. Four years later a second severe but single blood-spitting. He continued to work for eight years in fair health. Then a series of attacks of bleeding, cough and expectoration, tubercle bacilli, and a rapidly developing tuberculosis.

The treatment is that of cardiac inadequacy, and the most important single factors in it are well-ordered rest and well-regulated exercise.

DISCUSSION.

Dr. J. M. ANDERS (Philadelphia, Pa.): The subject of blood-spitting and cardiac diseases is an important one. Difficulties in diagnosis have been referred to, and I wish to emphasize particularly the difficulty that I have met with in a few cases in differentiating aortic insufficiency associated with hæmorrhage of the lungs from pulmonary tuberculosis. It is true that the cases of aortic insufficiency associated with pulmonary hæmorrhage form but a small percentage of the total. On account of certain symptoms, such as flushes and night-sweats leading to exhaustion, and blood-spitting, all of which may arise in aortic regurgitation, this disease may bear a close resemblance to pulmonary tuberculosis. I agree with Dr. Wilson that the incidence of blood-spitting in cardiac diseases is

greater than has been supposed. In the treatment, also, I fully agree that no special form of medication should be instituted, except in the very few instances in which it is decidedly exhausting in its effects.

Dr. De Lancey Rochester (Buffalo, N.Y.): This matter of blood-spitting in heart disease always interests me greatly, and I rise just to emphasize the importance of the distinction made, and also to state that I do not think that we can always positively say that it is due to the heart disease alone without further inspection of the lung. I call to mind one case, exceedingly instructive, of a young lad in the General Hospital in Buffalo, who came in with rheumatism, with an endocarditis, mitral stenosis, and aortic insufficiency combined—a combination which I have rarely seen. He came in spitting blood, and no tubercle bacilli were found in his sputum. He improved decidedly, and finally got up and was moving about the ward. He was suddenly taken with severe pain and hæmorrhages, and he had all the signs of a pulmonary infarction. Tubercle bacilli appeared in his sputum. There was a case which would undoubtedly have gone without appreciation of involvement of the lung except that he was under observation. The boy finally died of tuberculosis. Unfortunately an autopsy was not allowed. There is one other thing I want to say about the treatment of hæmorrhage. Within the last two years there has been brought forward in Buffalo through the studies of Dr. Busch and Dr. Clowes (Dr. Busch being the Professor of Physiology and Dr. Clowes Assistant in the New York State Cancer Laboratory) a means of treatment of hæmorrhage which has proved very successful in various cases. This is the production of a serum from which the anti-thrombine, which is normally in the blood, is removed by their method of procedure. I will not go into detail, because a paper will be published in the *New York State Medical Journal* in regard to this matter. About 15 c.c. of the solution is made, and is injected subcutaneously, or in some cases intravenously, I think, which has as good results in one way or the other. These injections stop hæmorrhage in many cases. In five cases, three of pulmonary tuberculosis and two of hæmorrhages of the stomach, the results have been remarkable, the hæmorrhage stopping within a few hours after the injection of the serum, and not recurring. Sometimes a number of injections are necessary, but I understand that this material will be put upon the market soon from one of the large manufacturing houses. Of course all hæmorrhages cannot be stopped with a serum, but I think it is well worth considering.

Dr. J. H. Elliott (Toronto, Canada): I would like to relate one case, a woman, aged about 36, who has had recurring hæmorrhages for some fifteen or sixteen years; they were supposed to be due to pulmonary tuberculosis. No tubercle bacilli had ever been found, but when I first examined her I found a fibrous condition of the right apex. The signs were slight, however. During the first two hæmorrhages I could find no physical signs in the lungs. On the third

occasion I was rather surprised to find the signs in the left apex, opposite to that which I had diagnosed formerly as tuberculous. At this time there was present, which I had not found before, a murmur of mitral regurgitation. The heart was somewhat enlarged; I had an X-ray made of the chest, and with the fluoroscope there was a pulsating tumour on the descending arch of the aorta, the side on which the physical signs of hæmorrhage had appeared.

Dr. J. C. Wilson: I will add to what I have said a few words. The importance of blood-spitting in heart disease depends upon two facts. First, that in the great majority of cases the quantity of blood lost is very small, and the hæmorrhage occurs at long intervals. Its symptomatic value may for this reason be wholly overlooked. Second, that hæmoptysis to many practitioners suggests disease of the lungs rather than disease of the heart. Hence uncertainties, even errors, in diagnosis. The difficulties in diagnosis are increased by the occasional occurrence of copious blood-spitting in heart disease and in associated cardiac disease and pulmonary tuberculosis. The necessity of a correct diagnosis in any given case arises from the fact that blood-spitting in heart disease tends to relieve congestion, and is often followed by relief of symptoms and does not usually call for energetic treatment; whereas blood-spitting of lung diseases usually demands active therapeutic measures, and is liable to be followed by fever, broncho-pneumonia and an extension of the original process.

CLIMATE OF THE SAN DIEGO (CALIFORNIA) REGION, WITH RELATION TO RENAL DISEASES.

By P. M. CARRINGTON, M.D.
Surgeon, U.S. Public Health Service.

One of the objects of this Association being the study c climatology, its founders were obviously of the opinion tha climate has an effect on the influence of disease, and therefore must have a therapeutic value in the treatment of disease.

Much of the literature of climate in recent years has dealt with the subject from the standpoint of the value, or lack of value, of climatic treatment of tuberculosis, but other diseases are affected (beneficially, some of us believe) by suitable climates, and among such diseases chronic nephritis in its various forms may be classed.

Most of us who extol the excellencies of the climate of some particular locality are, in a measure at least, prejudiced, either by personal benefit received, pardonable local pride, or personal interest. The writer belongs to the first class, having recovered from a tubercular infection in New Mexico, and afterward having derived great benefit from a two years' residence in San Diego, where he was sent to recuperate from an attack of nephritis.

Our opinions as to the value of this or that climate are largely empirical, being based chiefly on clinical experiences. Still, a study of mortality statistics throws some light on the subject, and if confirmatory, strengthens our convictions and fixes our opinions. It has been said that

figures do not lie, but that liars sometimes use figures; without accusing our statistics of mendacity, or acknowledging myself mendacious, it may be said that in considering mortality statistics with reference particularly to the chronic diseases, some corrections or explanations are necessary to make them tell the truth; for in our system of collecting vital statistics no provision is made for charging back to the locality of origin deaths of transient residents, from diseases contracted elsewhere than at the place of death, so that in the south-west, for instance, where large numbers of consumptives resort, the death-rate from tuberculosis is high, although the climate of that region is a most favourable one for consumptives.

Geographical and General.

San Diego, California, at the present time a city of between 75,000 and 100,000 population, is located on the shores of the bay of the same name and the Pacific Ocean, in north latitude about 32·5°, about seventeen miles north of the Mexican border.

That portion of the town along the bay and ocean front is but little elevated above sea-level, but the main portion of the residential section is built on hills and mesas, ranging from one to two hundred feet in elevation, and separated by many cañons leading into the bay, thus giving the city a most excellent natural drainage.

The character of the soil is such (being composed largely of decomposed granite on hills and mesas, and sandy loam in the valleys), and the drainage so excellent, there is practically never standing water or mud even after the heaviest rains. Fifteen miles inland we strike the foothills, and a little farther east the southern part of the Sierras, which in this latitude rise to an altitude of fifteen hundred or two thousand feet. In this region flowers blossom every month

in the year, lawns are ever green, and these add beauty to the scene and promote enjoyment and pleasure. It is the land of the fig, the lemon, and other citrus fruits.

All sorts of fruits, berries and vegetables thrive during the greater part of the year, and the adjacent waters supply a variety of edible fish, all very important factors in the dietary of nephritics; and the city is furnished with an ample, unfailing supply of pure mountain water, while an excellent sewage system assists in maintaining the exceptionally good sanitary condition of the municipality. The entire county of San Diego is traversed by a recently completed system of good roads, built at an outlay of $1,250,000.

History of Weather Observations and some Opinions of the Climate.

The Government has maintained uninterrupted meteorological observations at San Diego since 1849, when medical officers of the army instituted them, and continued in charge of them for over twenty years. In 1871 these duties were taken over by the Signal Corps of the Army, which had charge of such matters until the Weather Bureau took charge of all meteorological stations in 1891.

For the past fifty years the meteorological instruments, although their location has been changed several times, have been located within a radius of half a mile, so that in estimating the character of the climate we have not only the advantage of long-continued observations, but also for the last fifty years observations made under practically unchanged conditions.

Non-instrumental weather observations, indeed, extend back to 1542, when Cabrillo entered San Diego Bay and recorded its excellent qualities as a harbour of refuge from the south-western gales, and Father Junipero Serra, who established the first California Mission at San Diego in 1769,

praised the climate, which he said reminded him of that of Spain.

Richard Henry Dana, in his "Two Years Before the Mast," published in 1835, gave an account of the early days of this region, and recorded his observations of the climatic conditions.

Alexander Agassiz visited San Diego in 1872, and said, speaking of the climate: "This is one of the favoured spots of the earth, and people will come to you from all quarters to live in your genial and healthful climate—a climate that has no equal."

General A. W. Greeley, while head of the Signal Service, U.S.A., wrote: "The American public is familiar on all sides with elaborate and detailed statements of the weather at a thousand-and-one resorts. If we may believe all we read in such reports, the temperature never reaches the 80's, the sky is flecked with just enough cloud to perfect the landscape, the breezes are always balmy, and the nights ever cool. There is possibly one place in the United States where such conditions obtain: a bit of country about forty miles square at the extreme south-western part of the United States, in which San Diego, California, is located."

General Characteristics of the Climate.

In general, the climate of San Diego is characterized by uniformity of temperature, the average daily range being about 10 to 12 degrees, and the variation between winter and summer being less than 20°. There is also a high percentage of sunshine, which is even more constant in winter than in summer. Very hot and very cold periods are infrequent, high winds are unknown, and fogs are of less frequent occurrence than in the average sea coast locality. The climate may be described as cool in summer and warm in winter, and the short range of the temperature is due partly

to the latitude, partly to proximity to the sea, and the absence of mountains in the immediate neighbourhood contributes to infrequency of cloud or fog.

In this latitude one would naturally expect very hot days in summer, but great heat is prevented by two factors: During the forenoon in summer a low-lying cloud, locally referred to as the high fog, but spoken of by the Spaniards as "El velo qui cubre la luz del sol," or "El velo de la luz," "The veil which covers the light of the sun," or "The veil of the light," tempers the heat of the day until the trade-winds blowing from the ocean dispel the cloud, which is no longer necessary because of the cool breeze.

The temperature goes as high as 90° four hours in a year, and as low as 40° for ten hours. The mean maximum ranges from 62° in January to 75° in August, and the mean minimum from 44° in January to 63° in August and September.

The annual rainfall of San Diego is ten inches, and falls during the so-called rainy season, which extends from October or November until March. The humidity is relatively high, ranging from 67 in December to 80 in July, with a mean annual of 75. Occasionally temperatures of 100° F. have been recorded. These high temperatures occur during the so-called desert winds, but at such times the relative humidity is very low, 10 per cent., or even as low as 3 per cent., having been recorded.

I will not bore you with statistical tables, but briefly, the extremes of temperature in San Diego are 101° and 32°; the average of the three consecutive warmest days was 82·9°, while the average of the three coldest days was 40·2°, recorded in September and January respectively. In a period of twenty-five years the thermometer was lower than 80° on an average of 364 days a year, while the average daily range of temperature during the same period was 13°, and

the average difference between the mean of one day and the next is 2°.

The sun shines in San Diego on an average of 356 days a year, and the percentage of possible sunshine is 68, and the equability of temperature is the characteristic of the climate to which your attention is particularly invited.

Mr. Ford A. Carpenter, a corresponding member of this Association, and for sixteen years in charge of the Weather Bureau at San Diego, summarizes the climate as follows:—*

"*Temperature.*—Since the beginning of meteorological records the temperature has averaged less than one hour per year above 90°. Highest and lowest temperatures ever recorded are 101° and 32°, and no snow has ever fallen, although the records extend back to 1871.

"*Rainfall.*—The annual rainfall averages 10 in. Back from the coast the rainfall increases to over 40 in. It is in this well-watered region that the magnificent water-supply of San Diego is located.

"*Wind.*—The sea breeze keeps San Diego cool in summer and warm in winter, and the near-by mountains and desert give it a dry marine climate. The wind averages five miles per hour throughout the year.

"*Sunshine.*—The sun shines in San Diego on an average of 356 days a year. The photographic sunshine recorder shows that for twenty years there has been less than nine days a year without one hour or more of sunshine.

"*Comparative Seasonal Temperatures.*—Temperatures are usually shown on a globe by lines which pass through regions of the same degree of heat or cold. Red lines of 60° or 70°, showing the summer temperature at San Diego,

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* "The Climate and Weather of San Diego, California," by Ford A. Carpenter.

NOTE.—The great California freeze occurred January 6-7, 1913, after the above was written; at this time the temperature at San Diego was lower than 32° for 12 hours, reaching an absolute minimum of 25°, the lowest ever recorded.

also enclose Alaska and Siberia. Blue lines of 50° and 60°, showing the winter temperature at San Diego, enclose Egypt and Arabia. Thus San Diego may be said to have Alaskan summers and Egyptian winters."

Climatic Prevalence of Nephritis.

Dickinson has shown, with relation to Great Britain and Wales, that deaths from renal diseases increase with the variability of the temperature in the various parts of the kingdom, ranging from 1 nephritis in 53 from all causes in Aberdeen, with an annual mean temperature of 47° and a mean monthly variation of 30·7°, to 1 in 487 in Shetland, where the mean annual temperature is 43·8° and the mean monthly variation 19·9°.

On the western coast, by reason of the influence of the Gulf stream, the winters are warmer and the summers cooler, and a fairly uniform temperature prevails throughout the year. On this coast diseases of the kidney are less frequent by one-half than on the eastern coast of England, bathed by the cold waters of the North Sea, where the summers are much hotter and the winters colder than on the Atlantic shore, and where there is a wider range of temperature and greater variability of climate, and he attributes the extreme infrequency of renal diseases in the Shetland Islands to the uniformity of the temperature, notwithstanding its comparative low range. He says :—

"Renal disease, putting aside that of lardaceous origin, is the compatriot of wheat and barley rather than of the vine and of the olive. It abounds wherever the climate, however cool during the winter, is warm enough in the summer, as in Canada, to bring wheat to perfection; and becomes scarce where oranges and lemons grow, and where deciduous trees as generally characteristic of the scenery are replaced by palms and other tropical endogens."

It may be stated that the figures given by Dickinson refer to the year 1863, when diagnosis was less accurate than at the present time.

Consulting a recent report of the Registrar-General of Vital Statistics for Great Britain and Wales, I find that renal diseases have greatly increased in that country in recent years, although their relative climatic prevalence remains essentially the same, while the mortality statistics of our own Census Bureau show that renal diseases cause a much larger percentage of the total deaths in this country than in Great Britain.

Our own vital statistics are less satisfying and convincing than those of Great Britain for several reasons. The registration area of the United States includes but little more than 50 per cent. of our entire population, and a considerable part of that percentage has been so recently added that, while the laws are excellent, they have not yet attained full perfection of execution and administration. Again, our people are less firmly attached to localities, or are more roving than our English cousins, and so mortality statistics of chronic diseases are less truly indicative of actual health conditions. The mortality rates for some sections thus show for some of the chronic diseases higher percentages than local conditions justify, by reason of the large numbers of invalids resorting to those localities.

If it ever becomes possible to require every physician to keep complete records of all cases of disease and injury treated, as hospitals now do, and make periodical reports to the health authorities, then we shall have correct and complete morbidity and mortality statistics.

In the registration area of the United States the percentage of deaths from nephritis to the total mortality for 1909 is one to fifteen and sixteen one-hundredths, this including both urban and rural rates. In New York City

the proportion is 1 to 11; Philadelphia, 1 to 10; St. Louis, 1 to 11·29; and, in general, it may be stated that the rate is high in cities having a very variable climate, subject to great and sudden changes.

Herrick, in Osler's "Modern Magazine," says:—

"The relative infrequency of this form of nephritis (chronic interstitial) in the warmer climates has been frequently noted. This may be in a measure accounted for by the fact that in the warmer climate there is less exposure to cold, inclement weather, and sudden changes of temperature."

He advises resort to a warmer climate in winter and to a cooler one in summer for nephritics.

Now, the proportion of deaths from nephritis in San Diego to the total from all causes is 1 in 18, but eliminating from consideration deaths from nephritis occurring in persons who had lived in San Diego less than twelve months, the proportion is 1 to 43. The Board of Health in San Diego has no data as to the length of residence in the cases which had been there more than twelve months before death, but based on experience of local physicians, few or none occur in residents. This, on the face of it, while establishing the fact that nephritis is not relatively of so frequent occurrence in the climate of San Diego as elsewhere, does not seem to prove the excellencies of the climate in the treatment of the disease, but we must consider that if one desires to be benefited by climatic treatment, one must resort to the desired climate before the disease has completely demolished the structure of the organ for which relief is sought, since no remedy, climatic or otherwise, is capable of restoring or replacing tissue which has already been destroyed, and it is the common experience of all climatic resorts that a vast majority of patients come after their organs are irreparably damaged.

Bullard, of Los Angeles, in his work on "Apparent and Actual Mortality," written in 1893, showed that 25 per cent. of those dying in Los Angeles from nephritis had come to California within two years in an advanced stage of the disease, and the experience of San Diego physicians shows that the percentage of imported cases is much larger in San Diego at the present time, and this is supported by the mortality statistics of the San Diego Health Office above referred to.

To state the matter in somewhat different form, the death-rate in the entire registration area, as shown by the census reports for 1909, was 1,440 per 100,000 of population; from Bright's disease 95·2, and from pneumonia and other diseases of the respiratory organs 178·5. During the same year in the entire State of California the corresponding figures were 1,342·8, 85·3 and 144·3. For the State of New York, 1,566·2, 127·5 and 226·8. For the City of San Diego, 1,594·2, 89·9 and 161·3. These are the uncorrected figures, no allowance being made for non-resident decedents. The rates for pneumonia and other respiratory diseases are given to show the comparative infrequency in San Diego of these diseases, which are often intercurrent cause of death in subjects of renal disease.

Figures of this character might be multiplied indefinitely, but enough have been quoted to show that climate does play a part in the causation of renal diseases, although I do not mean in any sense to minimize the other well-known causes, nor even to claim that unfavourable climatic conditions may have a preponderance of causative action.

Advantages of the Region for this Class of Diseases.

The first and chief advantage of this climate in renal disease lies in its equability and uniformity, not only as between night and day, but as between the different seasons, there being no sudden drops in the temperature, and the

difference between summer and winter temperatures being much less than is frequently experienced in the Mississippi Valley and on the Atlantic coast in a period of twenty-four hours. As a consequence of this uniformity and equability there is a relative infrequency of the acute respiratory and intestinal diseases, which so often in less favourable climates become the intercurrent cause of death in nephritis.

Since the climate is one suitable for and favourable to nephritics during the entire year, it becomes possible for these patients to remove with their families to this region, establish homes and settle permanently, thus avoiding the separation from family and homesickness, which are so often inseparable from climatic resorts.

While the climate of San Diego is such as I have described, there are two other distinct climates different from that of the immediate coast within a distance of forty or fifty miles from the City of San Diego. One is the climate of the mountainous district some twenty miles inland, which is characterized by warm summers and cool winters, with a considerable daily range of temperature, moderate daylight rains, low relative humidity, fairly heavy winter rains, and some thunderstorms during the summer.

Another twenty miles eastward we have a distinct desert climate, with its almost unvarying sunshine, warm winters, and with great variation between day and night temperatures, hot summer days, extremely low relative humidity, and almost no rainfall.

So persons resorting to the San Diego climate may readily and easily avail themselves of a temporary change without travelling great distances, and at trifling expense.

Reference to some San Diego Cases.

My friend, Dr. P. C. Remondino, for nearly thirty years a resident of San Diego, and who has written very extensively concerning the climatology of Southern California,

says: "A well man will stand less chance of ever becoming sick here, and an invalid will, on the average, live longer and more comfortably and with the greatest possible and probable chances of ultimate recovery than in any other portion of the United States."

Two of the prominent physicians of San Diego went there on account of nephritis, one sixteen and the other six years ago.

The first was apparently in a very advanced stage of the disease, with general anasarca, ascites and great general debility, and was physically unfit for the least activity. In such cases we must use the expression "cured" with great caution, but this gentleman is now doing a very large practice and is very active in many ways, and is to all appearances, intents and purposes a well man.

Another very pronounced case is that of a gentleman sent to San Diego sixteen years ago on account of nephritis, and, as his medical adviser said to me, "completely water-logged," and who for several years has taken a very active part in the political life of the community; he is to-day a vigorous and apparently healthy man. These are but a few examples of the scores of well-known citizens whose cases are equally striking.

It is a climate which necessitates no seasonal change of clothing, where neither great heating nor cooling is required at any season, where vegetables and some fruits grow during the entire year, making proper diet for nephritics easily obtainable. In such a climate attacks of diseases invited or influenced by change of weather, diet or clothing, must necessarily be reduced to a minimum, and Dr. Remondino says:—

"I know from observation that a weak heart and defective kidney will support work better here than in the East."

Conclusion.—Experience shows, and opinions of those

whose judgment we must respect support the proposition, that renal diseases are more prevalent in climates subject to great and sudden changes of temperature and where other causative factors are practically the same as in regions where such diseases are less prevalent. Vital statistics, while not so conclusive, in general bear witness to the same facts.

The experience and testimony of our *confrères* in localities of equable climate is to the effect that nephritics do well, feel better, live longer, and even in some cases seemingly recover in climates of which San Diego presents an example. That a part of the observed beneficial effect may be due to more sane modes of living is admitted, but both reason and experience persuade us that the even climate must be credited with an influence in the general good results, even though the exact proportion of its influence is not demonstrable.

DISCUSSION.

Dr. F. M. POTTENGER: I would like to say just a word regarding this paper of Dr. Carrington. I think he has described to you this morning the climate of that particular part of the western coast which is most suitable in nephritic cases. On the western coast there are great differences in climatic conditions in places which are comparatively close together. For example, San Diego, Los Angeles, Santa Barbara, and San Francisco all have distinctly different climates, and yet they are all on the coast and all within six hundred miles of each other. San Diego has a peculiarly equable climate, therefore it is particularly suited to nephritic cases which require climatic conditions free from rapid changes. The daily variation in San Diego, as mentioned, I believe, by Dr. Carrington, is ten degrees between day and night. Our variation at Monrovia, which is 120 miles from San Diego, is thirty degrees between day and night. Los Angeles, which is twenty miles from Monrovia, has a difference of twenty degrees between day and night. At my institution we have found that by taking the temperature at seven o'clock in the morning and adding thirty degrees we have the approximate maximum temperature for that day. Now, regarding climatic change, we have learned a great deal

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NOTE.—For the meteorological data and figures in this article I am indebted to Mr. Carpenter's work, "The Climate and Weather of San Diego, California."

in recent years which makes climatic change less necessary in the handling of many cases which were previously handled in this way alone. We have not, however, reached the point where it is less desirable. It is much easier for patients to live under climatic conditions which are suitable to them, conditions which make life easy and comfortable, than under unfavourable climatic conditions. Patients suffering from nephritis can be sent to a place like San Diego, where they can live out of doors all the time. They are invited to be out of doors by the beautiful surroundings, and the conditions present are conducive to a happy life, which helps them to bear the restraint which is necessary for them to follow in order to give themselves the best chance of life.

Dr. A. C. GETCHELL: I think this is a very important matter, and I am free to say that many of the troubles which my patients at home have, from time to time, are due to the effects of the climate in which they live, and I hope that those who live in these better climates will continue to impress upon us their importance in the treatment of pulmonary tuberculosis. It is true that most of our patients must make the best of unfavourable climates because they cannot afford to leave them for better ones. There is a growing tendency to put more and more stress on the minute regulation of life, diet, hygiene, and other conditions, acting on the body every minute of the day, which tend to make the body a perfect biological laboratory. If this is so it is also true that the quality of the atmosphere lived in and breathed is an important factor in the treatment of the disease, and the value of climate cannot be too persistently urged upon us.

Dr. CASSELBERRY: A brief sojourn at San Diego some years ago tends to confirm its climatic advantages as described by Dr. Carrington. There is not only equability of temperature, but the general quietude is conducive to equanimity of mind, with a disposition to get out into the sunshine, and but little disposition to do anything else.

Dr. E. FLETCHER INGALS (Chicago, Ill.): I heartily agree with the gentlemen who are impressed with the advantages of climate, but it appears to me that we are liable to draw false deductions here as well as to reference of use of drugs and some other things as was demonstrated in the discussion which just preceded these papers. I was in San Diego a few years ago in July. I carried my overcoat with me about half of the time, and wore it the other half of the time, and an old physician, a man who had been there for many years, told me it was the worst place in the world for bronchitis, and I think his observation ought to be of some value at any rate. I heartily agree with the paper, but there was another inference that might be drawn from two of the statements—one that the great changes in climate, or the great changes in the climatic conditions, perhaps, during the day accounts for many of the cases of renal trouble, and that more people died of renal trouble who had only been there twelve

months than in the case of those who had been there much longer. The inference would be that the climate took them off pretty rapidly.

Dr. CARRINGTON: Parts of my paper were not read because the Chairman warned me that I only had two minutes' time left, whereas I had about ten minutes' more reading to do. The difference in the climate referred to by Dr. Pottenger is brought out in the paper in the part that I did not read by the statement that there are three distinct climates within a range of fifty miles in that one county. Of course, a vast majority of people suffering from any given disease must have their treatment at home; we know that, but if climatic treatment adds a little bit to my chances, if I have this or that disease, then I want the benefit of that little percentage whatever it may be, and those of us who can have the benefit of climatic treatment ought to have it, though the percentage of benefit may be small. Now as to those people who died within a year; that, I think, I covered by the general statement that they are practically dead when they come. Those people come in the vain hope that benefit will be derived. Had they come a few years sooner they would have been living many years yet.

MINERAL SPRINGS; THEIR ANALYSES, CLASSIFICATION, THERAPEUTIC USES, RADIOACTIVITY, AND NEWER METHODS OF APPLICATION; WITH SPECIAL REFERENCE TO AMERICAN SPRINGS.

By GUY HINSDALE, A.M., M.D.
HOT SPRINGS, VIRGINIA.

The modern student and practitioner is confronted with the most difficult and abstruse questions relating to the chemistry and physiological action of mineral springs, questions that are almost the despair of one of only ordinary scientific training. The chemical analyses of waters on which so much stress has been laid ever since analytical chemistry has been employed to determine their character are now themselves in process of passing into history. A glance at recent publications of the chemical division of the United States Geological Survey shows how completely the modern chemist has changed his notation to conform with the latest discoveries in the science of molecules, radicles, atoms and ions.*

In what form and in what combinations the simplest salts

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* See Chase Palmer, the "Geochemical Interpretation of Water Analysis." *Bulletin* 479, United States Geological Survey, Washington, 1911. H. P. Talbot and A. A. Blanchard, "The Electrolytic Dissociation Theory," Macmillan Company, New York, 1905.

exist in mineral waters or in artificial solutions demands the most serious attention. Take, for example, pure water, and add to it sodium chloride and potassium sulphate. We have instantly formed in solution sodium sulphate and potassium chloride, in addition to the two original salts, as well as the sodium, potassium, chloride, and sulphate ions; so the chemists tell us. The electrolytic dissociation theory goes farther, and gives us an entirely new conception of the relation or radicles or ions in various solutions.

An inkling of the state of the radicles of salts in solution is given in what analytical chemists in later years have been stating as the "*hypothetical* combinations" appended to their analyses, so as to correlate them with the time-honoured method of notation.

In the past we have been furnished with data of the most diverse kinds. The late Dr. Crook, after an inspection of about 1,000 analyses, noted that at least forty-two methods of stating the results have been employed. They are all now likely to be superseded by the ionic form which assigns weight directly to the chemically active parts of the dissolved substances, instead of using those parts to build imaginary structures (Chase Palmer). The sooner we adopt a value of milligrams per litre, or parts per million, discarding the historic "gallon," and think only of radicles instead of salts, we shall make a great advance and put ourselves in harmony with modern chemical nomenclature.

For a comparison of these methods take the analysis of sea-water, a mineral water of the widest distribution.*

An ion is an atom or group of atoms, forming in itself a complete individual, and possessing a charge of electricity; they may be produced by the dissociation of larger electrically neutral molecules (that is, of the molecules of compounds

* See following page.

as ordinarily expressed by their chemical formulas) into smaller ones, bearing, respectively, equivalent amounts of positive and negative electricity. In every solution the aggregate of charges on the positive ions which it contains must be exactly equalized by the sum of the charges on the negative ions present, otherwise the solution as a whole could not be electrically neutral. There is, apparently, in solutions some specific power in the solvent by virtue of which it is able to so force itself in between the parts of the molecules as to separate them into ions. Different solvents vary greatly in the extent to which they possess this power, water ranking as the most powerful. (See Talbot and Blanchard.)

OCEAN WATER.

AMOUNTS ASSIGNED TO HYPOTHETICAL COMBINATIONS			AMOUNTS ASSIGNED TO RADICLES	
Combinations	Milligrams per litre	Grains per gallon U.S.	Radicles	Milligrams per litre
Sodium chloride ($NaCl$)	27,215	1,868	Sodium (Na) ..	10,710
Magnesium chloride ($MgCl_2$) ...	3,807	220	Potassium (K) ..	390
Magnesium sulphate ($MgSO_4$) ...	1,658	96	Magnesium (Mg) ..	1,300
Calcium sulphate ($CaSO_4$) ...	1,260	73	Calcium (Ca) ...	420
Potassium sulphate (K_2SO_4) ...	863	50	Sulphate (SO_4) ...	2,700
Magnesium bromide ($MgBr_2$) ...	76	4·4	Chloride (Cl) ...	19,350
Calcium carbonate ($CaCO_3$) ...	121	7	Bromide (Br) ...	60
			Carbonate (CO_3) ..	70
Total	35,000	2,318·4	Total	35,000

Dr. R. Fortescue Fox, of London, presented this subject clearly in his Hyde Lectures before The Royal Society of Medicine last year.

"In all medicinal springs no doubt much importance attaches to the form in which the ingredients are presented in a more or less dilute solution. It would even seem that very dilute waters are sometimes the most active. Water

has the power of fluidifying or rendering motile the majority of solids. This fluidification, as in the case of vaporization, is associated with a loosening of the mechanical and chemical structure of the particles. This process, as applied to salts, is described as dissociation. Water further has the property of taking up molecules produced in this manner between its own molecules, rendering them fluid and motile, dissolving them and increasing their chemical activity.

"Moreover, these separated particles, or ions, whether bases or acids, carry a positive or negative electric charge, and do not exhibit the usual properties of the free elements. In a solution of sodium chloride, for instance, completely ionized, neither the kathion (sodium) nor the anion (chlorine) exhibits the properties of these elements in the free state. It is noteworthy that the degree of ionization is in proportion to the dilution of the solution. In many medicinal waters it is supposed that saline constituents are in a state of complete ionization."

We now have some insight into the remarkable effect of waters of low mineralization, and can understand why the free motility of the particles of matter may be essential to active metabolic processes, as had been suggested.

All waters carrying less than 150 parts per million* of total mineral matter are considered "low"; over 500 parts

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* Equivalents for converting analyses of water into parts per million:—

Parts per	100,000 × ·7	=	grains per imperial gallon (277 cubic inches)
,, ,,	1,000,000 × ·07	=	,, ,, ,, ,, ,,
,, ,,	1,000,000 × ·058	=	,, ,, U.S. ,, (231 cubic inches)
Grains per imperial gallon	÷ 0·7	=	parts per 100,000
,, ,, ,,	÷ 0·07	=	,, ,, 1,000,000
,, ,, U.S. ,,	÷ 0·58	=	,, ,, 100,000
,, ,, ,, ,,	÷ 0·058	=	,, ,, 1,000,000

One part per hundred (percentage)	=	10,000 parts per million
,, ,, ,, thousand	=	1,000 ,, ,, ,,
,, grain per litre	=	1,000 ,, ,, ,,
,, milligram per litre	=	1 part ,, ,,
,, grain per U.S. gallon	=	17,118 parts ,, ,,
,, ,, ,, imperial gallon	=	14,261 ,, ,, ,,

"high"; and over 2,000 parts "very high." Of course, low concentrations of powerful radicles like arsenic may be therapeutically very powerful, or even toxic.

Once firmly established in the mind the ionic form of expression stated in parts per million, or milligrams per kilogram, will be found the easiest to comprehend and to calculate and in harmony with what we hope will be the universal methods of stating the composition of waters.

The classification of mineral waters has been attempted by many men of many minds, but we believe we are indebted to Dr. A. C. Peale, of the Smithsonian Institution, Washington, for the best and most comprehensive classification, first presented in a paper read before the American Climatological Association in 1887. He has classified all the mineral springs of the United States that have been analysed, and he has found it to meet the requirements in every case.

All mineral waters are divided into two groups, and are then treated precisely alike with reference to their chemical contents. Group A, *cold* (non-thermal) *springs*. Group B, *thermal springs*. A spring is "thermal" when its temperature is above the mean annual temperature of its locality; it is "cold" when below that temperature. A spring, therefore, might be thermal in Alaska, when a spring having an identical temperature would be cold in Virginia.

Class 1, alkaline	Sulphated Borated Muriated
Class 2, alkaline-saline	Sulphated Borated Muriated
Class 3, saline...	Sulphated Muriated
Class 4, acid	Sulphated Muriated Silicious
Class 5, neutral or indifferent waters	

The existence or non-existence of gaseous contents may be shown thus:—

(1) Non-gaseous.
(2) Carbonated (containing CO_2).
(3) Sulphuretted (containing H_2S).
(4) Azotized (having nitrogen).
(5) Carburetted (having carburetted hydrogen).

To the foregoing we must now add :—

(6) Radio-active waters.

Class 1. *Alkaline Waters.*—These may be termed sodic-magnesic, alkaline, chalybeate or calcic. Alkalinity may be due to sodium, potassium or lithium (primary alkalinity); or it may be due to the presence of alkaline earths, calcium and magnesium (secondary alkalinity). The term "primary," in this connection, indicates an association with the alkalies of the oldest rock formations, of which the alkalies are the principal soluble decomposition products. The alkalinity of waters due to contained calcium and magnesium is termed "secondary" because they occur in connection with rocks of more recent or secondary (stratified or sedimentary) formation. Springs of this class commonly contain carbonic acid gas, which increases palatability and solvent power; they also merge by gradation into the succeeding classes, alkaline-saline and saline. Some of the notable alkaline springs in the United States are: The Navajo spring at Manitou, Colorado; Ukiah Vichy, California, and California seltzer; Waukesha Springs (White Rock and Clysmic), Wisconsin; Londonderry Spring, New Hampshire.

These waters stimulate the action of the stomach and check acidity and fermentation; they increase the urine and lower its acidity, and have a good influence on the mucous membrane of the entire digestive tract. Hence they are used in the treatment of acid dyspepsia and flatulence. They are useful in chronic gastric catarrh with hyperchlorhydria, rheumatism and gout.

Diabetes was at one time supposed to be favourably influenced by the alkaline waters, and even still patients suffering from this complaint are commonly sent to La Bourboule, Vichy, or to Carlsbad. This mode of treatment arose from the view put forward by Mailke that the disease was due to incomplete oxidation of sugar owing to lack of alkali in the blood. This doctrine has long passed away, but the practice founded on it still survives. Many patients are, in reality, much benefited by a course at Vichy, at Carlsbad, or at other spas, but there is every reason to believe that this is due not to the waters, but to the careful *régime* and regulation of the diet. The alkaline salts have been shown by many observers to have no power to diminish the formation of sugar, while in all cases the copious administration of water is injurious. Unless alkalies are called for on some independent ground they should not be given in diabetes (W. R. Huggard).*

It used to be believed, and spring owners never failed to claim, that there were wonderful virtues in the lithium waters of this class. The advertised analyses showed as high as 22 gr. per gallon (379 parts per million) of carbonate of lithium in one case, and absolutely fictitious amounts in many cases. The U.S. Government has put a stop to this, and the word "lithia" does not now appear on so many American mineral water labels as formerly.†

It is not proved that lithia has any therapeutic effect very different from its congeners, sodium and potassium.

The waters of Carlsbad, in Austria, Fachingen, in Germany, and Vichy, in France, are notable examples of the alkaline waters.

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* See also von Noorden, "New Aspects of Diabetes," E. B. Treat and Co. New York, 1912.

† See author's article on mineral springs, Hare's "Modern Treatment," Philadelphia, 1911.

Calcic Magnesic Alkaline Waters.—The waters of secondary alkalinity, or those containing notable amounts of calcium and magnesium, are commonly denominated calcareous waters. They contain a notable proportion of the alkaline earths belonging to the secondary geologic formations. The salts of calcium are sparingly soluble in water, and give it a characteristic usually termed "hardness." Waters of excessive hardness are entirely unsuitable for drinking, but this quality does not prevent their use for bathing. Most of the thermal springs of Virginia and West Virginia belong to this class, the strongest or hardest being used for the baths, and those of weaker character being used internally. The Warm Springs, Hot Springs, and Healing Springs of Virginia are in the same valley where limestone belonging to the Cambrian, Trenton and Hudson (valley limestone) formations underlie the surface. These thermal springs range in temperature from 84° to 106° F., and have been used since the days of the American Revolution for bathing and internal use. The Healing Springs water, which is principally used internally, contains calcium bicarbonate as its principal mineral, analyses showing 356 parts per million, or 356 mg. per kilogram (20·7 gr. per gallon). Calcium sulphate is absent, and this is a great advantage, as indigestion and constipation are less likely to be caused. In this respect Healing Springs water bears a close resemblance to the waters of Wildungen. The waters used for bathing, those coming from the Boiler spring at Hot Springs, are considered too hard for internal use.

One of the notable discoveries of the last century was made by Thomas Clark, of England, who, in 1841, patented a process for removing the objectionable constituents of hard water. He discovered that ordinary lime water precipitated the soluble calcium bicarbonate as calcium carbonate, and that all the calcium was removed simultaneously from the

water so treated, and from the reagent as well. This process has been of immense value industrially and hygienically and has been widely employed.*

Ocean water contains an excessive amount of calcium and magnesium, not altogether dissociated from the chlorine, amounting in all to 420 parts per million for calcium, and 1,300 parts for magnesium; the carbonate content in this case is 70, the chlorine content amounting to 19,350.

Calcic, or calcareous, waters when not associated with chlorine or the sulphate radicle, have a beneficial effect in cases of gout and rheumatism and in counteracting hyperacidity of the stomach and urinary tracts. The calcium salts are excreted by the intestine in the form of a phosphate, and so the latter's transference to the kidneys and urine is prevented. Von Noorden has shown that the total amount of phosphates in the urine is diminished, and as the reaction of the urine approaches alkalinity, the proportion of disodium to monosodium phosphates is modified in favour of the former. Most of the waters contain, in the natural state, carbonic acid gas, and therefore are best used at their source. Calcium salts are eagerly sought by the acids of imperfect digestion. The latter readily combine with the bases thus furnished, and probably in this manner obviate union with similar bases found in bone, cartilage of joints, and in other natural tissues of the body. A proper selection of food is usually enjoined at spas for treatment of rheumatism and gout, in which vegetables rich in salts are recommended, so that diet goes hand in hand with mineral waters in the treatment of these affections.

Calcic and magnesic waters favourably influence rheumatism of a subacute or chronic type, pyelitis from kidney concretion, chronic gonorrhœa, and bronchial catarrh. They

* See Chase Palmer, *Bulletin* 479, *United States Geological Survey*.

probably also favourably affect scrofulosis, rachitis, and osteomalacia, as they supply the deficient salts.

Very recently attention has again been called to the need of lime salts in the treatment of tuberculosis. It is claimed that lime starvation is characteristic of these cases, and should be corrected by dietetic methods; in this connection calcareous waters should have a prominent place.*

There are valuable thermal calcareous springs in the mountains of Virginia, West Virginia, and Arkansas, some of them almost entirely free from sulphuretted hydrogen. There are valuable cold sulphuretted calcic-magnesic sulphated alkaline-saline waters at Richfield and Sharon, New York. The waters of Bedford Springs, Pennsylvania, of Contrexéville, France, and Leuk, Switzerland, are non-gaseous calcic sulphated waters.

Saline Waters.—This large and important class presents all gradations from the weaker alkaline salines to the strong brines. They may contain large quantities of hydrogen sulphide (H_2S) or carbon dioxide (CO_2). Like the alkaline waters, they may be referred to as having primary salinity (NaK), or secondary salinity due to the presence of the calcium and magnesium (earths) and the stronger acid radicles. The term "tertiary salinity" is used to represent acid waters in which the percentage values of the strong acids present are greater than those of the alkalies and earths.

Well-known examples of saline waters are those of Mount Clemens, Michigan; Glen Springs, Saratoga Springs, Clifton Springs, Westfield and Columbia Springs, New York; Caledonia Springs, between Montreal and Ottawa, Canada; Byron Springs, Eureka Springs and Calistoga Springs, California. These show a large amount of chlorides, and

* See van Gieson, *Transactions* of National Association for the Study and Prevention of Tuberculosis, Washington, 1912.

are therefore called "muriated salines." Another large division is called "sulphated saline," because of the predominance of the sulphate radicle. They contain magnesium sulphate (Epsom salt) and sodium sulphate (Glauber's salt), or their respective radicles. Other bases, such as calcium and iron, are also commonly associated; likewise large quantities of hydrogen sulphide.

The salines have an important place therapeutically on account of their laxative or purgative properties. They have an immense commercial value, and although in the United States there are large numbers of these springs, whose waters have been extensively sold for a hundred years, we nevertheless import vast quantities from Europe. The importation and sale of saline waters of the sulphated variety includes the well-known Hunyadi, Apenta, Rubinat, Carabaña, Seidlitz and Friedrichshall.

It should be noted that in the Saratoga waters, for example, there is more lithium than in so-called lithia waters; the preponderance of the sodium chloride, however, determines the character of Saratoga waters, such as Congress and Hathorn, as saline, and they are used accordingly. The chloride of magnesium in saline waters is a constituent of the Mütterlage of salt works; it is present in the Dead Sea and in the stronger brines of Michigan and New York. Potassium chloride and calcium chloride are of no great advantage as such when taken internally, although the former is credited with increasing the flow of bile and improving the appetite. It is doubtful if we can differentiate any effect from these lesser constituents when using waters in which other elements greatly preponderate.

The use of sodium chloride waters undoubtedly causes an increase in the flow of gastric juice, bile, pancreatic juice and intestinal fluid; the appetite is usually increased, and the whole digestive tract freshened. There is little oppor-

tunity for intestinal putrefaction, fermentation or auto-intoxication from the alimentary canal when these waters are faithfully and judiciously used. The chlorinated waters have some advantages over the sulphated waters, or bitter waters, in the case of thin persons whose portal circulatory system is supposed to be sluggish. They correct atonic dyspepsia, furred tongue, bad taste in the mouth, and, as noted, almost always improve the appetite. This is the secret of the great popularity of Saratoga springs in the earlier days; those who went there to drink the waters were well rewarded, and we trust that under the State's ownership we may see that spa restored to its former sphere of usefulness.

The waters of French Lick, Indiana, in their artificially concentrated form, are likewise purgative, and depend on the presence of sulphates.

Saline waters have long been employed externally as baths for rheumatic and gouty affections; in connection with carbon dioxide they are used very extensively for the treatment of cardiac diseases. The plan of treatment afforded at Nauheim, in Germany, where the brothers Schott met with remarkable success, has been adopted at other European spas, and has been imitated with more or less success by artificial means. The great advantage which Nauheim has lies in the great quantity of flowing water containing carbonic acid gas in connection with chlorides and in the proximity of the salt works, where a strong brine or Mütterlage is available for use in baths. Last, but not least, the physicians of the place have developed a plan of treatment suited to a large number of patients presenting a group of symptoms of cardiac and circulatory disturbances. Constant experience in a given class of cases is a great aid in adapting the treatment to individual needs.*

* The system of Nauheim baths and resistant exercises is discussed more fully in the author's "Hydrotherapy," W. B. Saunders and Co., Philadelphia, 1910, and in W. Bezly Thorne's "The Schott Methods in the Treatment of Chronic Diseases of the Heart," fourth edition, London, 1902.

Schlemmer claims that too rapid hypochloruration and hyperchloruration (French terms for the effect produced by a diminution or excess of sodium chloride in the economy) occasion in the animal organism, besides other functional disturbances, cellular alterations which, in the first instance, are characterized by mucoid swelling of protoplasm; and in the second case, by atrophic lesions affecting the nuclear substance. These two procedures, carried on with moderation so as to avoid too sudden variations of osmotic equilibrium, enable one to make comparative studies, in the two cases, of electrical conductivity, of the effect of different poisons, and the influence of various infections. Experiments seem to show that, in general, organic resistance is greatly lessened in animals previously supplied with an excess of chlorides, by reason of lesions due to an overload imposed on cells by the fact of a stimulation of dialysis and consequent mineral disintegration. Schlemmer believes that we can compare the experimental data from the case of lesions caused by treatment not sufficiently gradual with the observed bad results following improper use of saline waters. The inference is made that waters are unsuitable for tuberculous patients. Saline baths, in the opinion of some of the German observers (Keller, Schlemmer), ought to be used with caution in tuberculous cases on the ground that they cause a more complete and intense combustion of the albuminoids in the system. They assume that saline waters used as baths increase the respiratory-urinary exchange for nitrogen and minerals, and that as tuberculous patients already present in general an increase of these metabolic processes, we ought to be cautious about adding to the tissue waste, otherwise the cure will be interrupted. They make an exception in the case of surgical tuberculosis. Their conclusion is that salt baths are counter-indicated in pulmonary tuberculosis.

In the case of inhalations of sulphurous waters or vapours which have had some vogue in France, the criticism has been made by M. Grehánt and Dr. Zaleske that sulphuretted hydrogen combines with the iron of the hæmoglobin, forming a compound similar to the exyhæmoglobin, and to that extent the hæmoglobin is lost to the organism; however, sulphurous waters may be useful in lesions of scrofulous type.

On the other hand, it is denied that hydrogen sulphide can combine with the iron of the hæmoglobin. The sulphur of sulphur waters is eliminated by the bronchial tubes, according to Claude Bernard, where it is combined with the different elements of the organism; but it is claimed that it does not act directly on the blood. I submitted this question to Dr. W. H. Howell, Professor of Physiology in Johns Hopkins University, and he corroborated the opinion that the hydrogen sulphide when inhaled would diminish the respiratory capacity of the blood. Gazaux has called attention to the favourable clinical experience in the Pyrenees in the use of sulphur inhalations practised there for a period extending over one hundred years. Dr. Albert Robin also refuses to condemn sulphurous waters in this connection, and incidentally makes a plea for the rehabilitation of chalybeate waters in tuberculosis, which have been under a ban for many years, having been originally condemned by Trousseau.

Iron, or Chalybeate Waters. Steel Waters of Europe.— Iron is found very frequently in mineral waters and in all of the four principal classes; we may have an alkaline chalybeate; an alkaline-saline sulphated chalybeate; an alkaline saline muriated chalybeate, and an acid sulphated or muriated chalybeate.

There are no absolutely pure chalybeate waters, but the

iron exists in connection with the bicarbonates, sulphates, chlorides, and with other metals, such as calcium, magnesium and aluminium. They are thermal or non-thermal, and are associated with free carbonic acid gas and hydrogen sulphide. A water containing bicarbonate of iron is usually preferable for continued use as a tonic to one holding sulphate of iron, as the latter is more astringent.

Free sulphuric acid and alum salts render some iron springs unfit for use; but where these constituents are in moderate quantity, and may be taken at their source, the effect is often beneficial. A relaxed mucous membrane with a tendency to diarrhœa and anæmia may be treated successfully with a water of this type. Good examples are Rockbridge alum water, Virginia; or the Oak Orchard Springs, New York.

For continued use in anæmia and chlorosis, we naturally choose a water with bicarbonate of iron from a cold spring with an abundance of carbonic acid gas. In the United States we have Berkeley Springs, West Virginia; "Round" Spring at Aurora Springs, Missouri. In Europe, those of Spa, in Belgium, Schwalbach, in Germany, and St. Moritz, in Switzerland, are famous. These waters contain respectively 109 parts, 95 and 47 parts per million (in each gallon 6·4, 5·6 and 2·8 gr.) of bicarbonate of iron. They are agreeable to take by reason of the large amount of carbonic acid gas present. English chalybeate spas include Tunbridge Wells, in Kent, and Flitwick Well, in Bedfordshire. The Flitwick water, one of the strongest iron waters in the world, contains 2,929 parts per million of sulphate of iron (170 gr. to the gallon), and is bottled for sale. The bottled water does not contain any carbonic acid gas. The waters of Harrogate and Buxton also contain iron; the so-called "Kissingen" Well, one of the eighty springs of Harrogate, contains about 170

parts per million of bicarbonate of iron, or 10 gr. per gallon; while the "Chloride of Iron" Well contains 227 parts per million (13·3 gr. of chloride of iron per gallon), and 191 parts per million (11·2 gr. per gallon) of bicarbonate of iron. The waters of these chalybeate springs of Harrogate are bottled after being artificially charged with carbonic acid gas. At Harrogate they are generally taken after being heated.

Waters containing bicarbonate of iron require great care in bottling, as most of the iron is liable to be deposited on the sides of the bottle in the form of an insoluble oxide. To prevent this the waters must be sterilized to prevent the growth of any microbes; they must be thoroughly charged with carbonic acid gas, and be very carefully closed. Chalybeate waters always give the best results when taken from the spring. One reason why the iron waters do not act so well after bottling is, that if much lime is present, as is frequently the case, the iron precipitates.

The waters of Schwalbach, on that account, keep better than those of Pyrmont, which contain lime in abundance. Those from the Prince de Condé, Spa, keep the best of all, and may be given in quantities from one to four tumblers a day. They aid digestion and improve the qualities of the blood.

Iron waters are best taken after meals in quantities varying from 4 to 8 oz. The use of 8 oz. of the weaker waters may produce headache in susceptible patients. It is best to order a saline water before breakfast when giving iron waters.

The waters of Franzensbad, Marienbad, and Kissingen (Rakoczy) all contain some bicarbonate of iron, and are bottled for use. Iron springs are rather common in the

United States. One of the purest chalybeate springs gives the following analysis :—

"Round" Spring, Aurora Springs, Missouri.

	Parts per million	Grains per U.S. gallon
Ferrous carbonate	87·8	5·13
Ferrous oxide	15·9	0·93
Magnesium chloride	118·9	6·95
Sodium chloride	68·6	4·01
Calcium sulphate	41·4	2·42
Lithia...	24·4	1·43

Iron Salts.

	Parts per million	Grains in one U.S. gallon of 231 cubic inches
Cresson Springs, Penna. Iron Spring	487·8	28·5
,, ,, ,, Alum ,, ...	763·4	44·6
Mardela Spring, Maryland...	196·8	11·5
Rock Enon Springs, Virginia	243·0	14·2
Massanetta, Virginia	53·0	3·1
Napa Soda Springs, California	135·2	7·9
Topeka Mineral Well, Kansas	481·0	28·0
Matchless Mineral Water, Alabama	17,973·0	1,050·0

Chalybeate waters are useful in anæmia, general debility and scrofula. The more astringent waters may be chosen in cases of chronic diarrhœa and in malarial cachexia. Iron waters are generally deemed unsuitable in any case of fever, in gastric irritability, and in pulmonary disease if hæmorrhage is a feature. Like most mineral waters they are diuretic. If associated with a little arsenic or manganese their value is probably enhanced.

Arsenic Waters.—Waters of this class are found in the

United States and Europe, the best known being as follows :—

	Parts per million	Grains of As per U.S. gallon
Mont Dore, Puy de Dôme, France ... .. La Bourboule	11·9	0·7
Levico, Austrian Tyrol, Strong	10·2	0·6
Roncegno	3·9	2·0
Crockett Arsenic Lithia Spring, Montgomery Co., Va.	2·4	0·140

These waters contain iron as sulphate. The small quantity of arsenic acid or arsenious salts is readily taken up in the blood-stream, and increases the amount of hæmoglobin. Arsenic has an effect on the skin which seems to be better nourished. According to the investigations of Schultz and Binz, the process of transformation of arsenious acid into arsenic within the body may possibly cause a to-and-fro movement of the oxygen atoms in the protoplasm, which, in connection with the direct effect of small doses of arsenic, stimulate the processes of tissue formation.

Gaseous Waters.—The presence of gases, such as nitrogen, carbon dioxide, or hydrogen sulphide adds a distinctive character to mineral waters, and sometimes suggests the name of the spring. It is a well-known fact that the estimation of gases in mineral waters is quite uncertain, varying more than in the analyses of solid contents by different chemists. Gases are, naturally, volatile, and must be estimated at the spring, and even then the results vary greatly. The fact that waters are released from all pressure on their emergence at the springs determines the escape of much of the contained gas. This has led to some very interesting questions, and, incidentally, to law-suits. These hinged on the point whether a water were still natural when

the original gas had escaped, and when the contained gas, or other similar gas, has been recombined with the water under artificial pressure. Natural waters, as well as artificial waters, are now subject to duty when imported into the United States, although natural waters were formerly admitted free. The famous case of the United States Government *v.* The Apollinaris Co., which arose in 1882, was decided after much litigation in favour of the latter.

It should be remembered that a given quantity of water, *e.g.*, 1 litre of water at 15° C., and at the ordinary atmospheric pressure, can contain the same same volume, 1 litre, of carbonic acid gas; but to contain five times that bulk it must be subjected to a pressure of five atmospheres. In the case of hydrogen sulphide at 15° C. and at atmospheric pressure as much as 3·24 volumes can be contained. Heating, however, in all cases speedily reduces the percentage of gas contained, and hence it is that cold gaseous springs are usually more highly charged than hot gaseous springs. Practically, all carbonated waters when bottled for sale are artificially charged. All mineral waters should be taken as fresh as possible. If bottled waters are used the best of corks should be used, and if this point is neglected the water is liable to deteriorate, lose its proper taste, and may be positively dangerous. Hence waters should be purchased from a large dealer whose stock is never allowed to deteriorate.

We should endeavour to cultivate a rational use of mineral waters, and a true knowledge of their contents and physiologic action. The majority of people depend on the advertised analyses of waters and the statements of their owners, or their agents, regarding their action on the system. Others rely upon personal experience at the fountain-head or on physicians who have studied their effects. The empiric method and the scientific investigation should go

hand in hand. The United States Government and the French Government, through the "Commission des Eaux Minérales" of the Académie de Médecine, have taken this matter in hand. For over ninety years no mineral water can be advertised as such, nor can a mineral water station be exploited in France without the favourable recommendation of the commission under whose direction the analyses are made. Such a guarantee of accuracy has been lacking in the United States until recently. It needs only a glance at the United States Government report on lithia waters to see what absurd claims were made by mineral spring owners as to the amount of lithia contained when the United States Government chemists showed that the lithia content in many cases was inappreciable.*

During the last five years a method of using natural mineral water hypodermically and intramuscularly has been developed in France. The late Dr. C. Fleig, of Montpellier, was an ardent advocate of the method and its chief exponent. Naturally a great many criticisms have been made of this procedure, but it is novel and worth looking to.†

This author had tried sea-water in tuberculosis cases and later made a remarkable study of the effects of mineral springs by hypodermic method. This included the sodium chloride waters of Balaruc, Homburg and Nauheim; the arsenical waters of La Bourboule, which contain also considerable sodium chloride; the stronger sulphuretted saline waters of Uriage; those of Kreuznach (Victoria Quelle); Perrière.

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* See *Bulletin* No. 91, Bureau of Chemistry, U.S. Dept. of Agriculture, Haywood and Smith. Lately the United States Government has undertaken a thorough investigation of the chemical and medicinal properties of the waters of Hot Springs, Arkansas, where there are two Government hospitals, and where the entire property, including the springs, belongs to the United States. This commission, involving an outlay of $50,000, will probably be authorized by the present Congress.

† C. Fleig, "Les Eaux minérales milieux vitaux. Sérothérapie artificielle et balnéothérapie tissulaire par leur injection dans l'organisme," pp. 513. Maloine, Paris, 1909.

All the waters which had a molecular concentration approximating that of the blood were injected in their natural state. The Balaruc water, for example, is absolutely isotonic with the blood; that of Homburg (Elizabeth Quelle), Kreuznach (Victoria Quelle), Nauheim (Karlsbrunnen), and Uriage are a little hypertonic, but can be injected without any inconvenience undiluted. The water of La Bourboule (Choussy-Perrière) is sufficiently hypotonic to be injected in its natural state up to 200 or 300 c.c. For the higher doses it is desirable to raise it to the isotonic state by the addition of sodium chloride.

Dr. Fleig, it appears, made the injections intramuscularly or hypodermically, the quantity used varying from 20 to 100 c.c. daily to 200 to 500 c.c. twice a week. The larger injections are given in the buttock; and, likewise, some of the smaller injections; but Fleig laid some stress on giving small injections near the local lesions such as ulcerated glands and fistulous tracks leading to bone. He used also wet sterile dressings of the water to be employed. When there are superficial or deep glandular lesions he attempted to bring on a direct local reaction with the water employed by making the injection in the neighbourhood of the lesion. He also used local injections of an ethereal solution of iodoform into the tuberculous glands if such are present. Care is taken in using gaseous waters to get rid of the excess of gas by agitating in a large flask.

The result to the patient is usually some reaction attended by chills, slight fever and sweats; they are not held to be toxic, but analogous to the symptoms produced by the larger injections of normal saline commonly employed. Fleig used his method intravenously as well as in the manner described. The condition of the patients reported on and the results are stated with considerable detail, and the outcome was favourable in most cases.

Radio-activity of Mineral Waters.

Not long after the discovery of radium by the Curies in 1898 the mineral springs of France were tested for radio-activity. Mme. Curie, Moureu, Laborde and Bertier had done the principal work, and they report most of the French springs radio-active. In Germany, those of Gastein, Karlsbad, Marienbad, Teplitz-Schonau, Fischau Baden, Joachimsthal, Odenwald, Pöstyen, Wiesbaden, and Kissingen are all pronounced radio-active by such observers as Mache, Meyer, Schmidt and Kurz, Jentzch and Heinrich. Ten years ago the waters of Bath, England, were shown to be radio-active by Professor John William Strutt (Baron Rayleigh). In the United States the chemists of the Geological Survey, Schlundt and Moore, have studied very thoroughly the mineral springs in Yellowstone National Park, and find the waters and gases in a very large number radio-active, far greater, as a rule, in this respect than any of the European springs. Professor B. B. Boltwood and Dr. Joseph Hyde Pratt, of Yale, have examined the Hot Springs of Arkansas, and found forty-four of these springs radio-active.

The measurements are all given in Dr. Boltwood's report, and he notes that the activity of the gas fell to half value in 3·9 days after removal from the source.*

These studies were made with Dr. Joseph Hyde Pratt at the direction of the Secretary of the Interior.

The method employed in determining the radio-active gas in water and the presence of radium salts in solution was by expressing the activity of the dissolved radium emanation in terms of the uranium equivalent; since the quantity of radium associated with a definite weight of uranium in a radio-active mineral has been shown to be perfectly definite

* *Amer. Journ. of Science*, August, 1905.

and unvarying quantity, this method of expressing the activity of a given quantity of emanation, as Dr. Boltwood shows, affords a correct and accurate standard for the comparison of samples of water from different sources. The same method and the same type of electroscope designed by Dr. Boltwood were used by Schlundt and Moore in the Yellowstone National Park in 1906.

All the springs of Hot Springs, Arkansas, are situated in a portion of a narrow valley about 500 yards long; they range in temperature from 35° C. to 64° C. (95° to 147° F.), and the total solids range from 170 to 310 parts per million, a rather low mineralization. The radio-activity, however, did not show any corresponding uniformity; the most active spring water showing over 500 times more than the least active. This was shown to be wholly independent of the method of collection and examination; neither did there seem to be any connection with the temperature, flow, location, or chemical composition of the waters of the springs and the observed differences in their radio-active properties. The second most radio-active water was found in one of the few cold springs on the reservation, and this seems to show that the thermal qualities of these waters and their radio-active qualities are due to quite independent causes.

There are different units of measurement of radio-activity which need some explanation. Curie, Laborde and Moureu took as a unit of the quantity of emanation the milligram-minute of bromide of radium; that is to say, of emanation discharged during one minute by a milligram of pure bromide of radium in solution. This unit is independent of measuring instruments, and is constant. It is called a "millicurie." A "curie" would therefore represent the action of 1 grm. of radium for one minute. The electrostatic unit is defined as being equal to the fall of tension, electroscopically, of one volt per hour, and by a litre of

A. Old Faithful Geyser, Upper Geyser Basin.
Yellowstone National Park, U.S.A.

B. Hot Spring, Norris Geyser Basin, where Thorium was first discovered in the United States. Testing radio-activity.

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radio-active water. It varies necessarily with the electroscope employed, and depends on the capacity of the instrument. It would appear that 116 units of volts equal one Maché unit, and 7,000 volts equal one milligram-minute unit of Curie. These relations seem at present to be generally accepted, but a uniform system ought certainly to be adopted to prevent confusion. The Radiological Congress at Brussels in 1911 formally adopted, at the suggestion of Löwenthal, the Curie unit.

One phase of this subject now assuming practical importance is the best means of rendering water radio-active for therapeutic purposes. Several forms of apparatus have been utilized to some extent in Europe. Three are of German origin, one is manufactured by the Radiogen-Gesellschaft of Charlottenburg, Berlin, another is French, and is known as the apparatus Armet de Lisle. In the "Radiogen" apparatus, water is made to pass through a radio-active substance; in the French apparatus, the water receives its properties directly from a small quantity of insoluble sulphate of radium, placed in a small cup of spun glass in a portion of the glass apparatus designed to receive the water to be treated. The radiogen apparatus yields a radio-activity in the neighbourhood of 3,500 to 4,400 volts; it is claimed by Jaboin and Beaudoin that the apparatus Armet de Lisle gives 11,000 to 13,000 volts, and that it is preferable because it renders the substances treated indefinitely radio-active.

The Radiogen-Gesellschaft offers also a drinking water in bottles of 20 c.c. The daily dose is three bottles. There are also capsules of radium bromide in physiological salt solution for subcutaneous or intramuscular injection, bath solutions, compresses, and "radiogen" mud for local application to diseased parts. This concern also equips emanatoria for the production of radio-active water and the inhalation of radium emanation. The electrostatic volts of these prepara-

tions are given by the makers, and are claimed to be constant. (See note, page 94.)

Another German preparation is on the market under the name of "Emananosol-Badtafeln," made by the Hochster Farbwerke. For these a value of 25,000 to 30,000 electrostatic units is claimed. Then there are the radio-active preparations of Kiel. These include bath and inhalation; the former are said to have 6,250 units, and the latter 6,000 to 12,000 units. As far as we know only the "radiogen" products are on sale to any extent in this country.*

Another method of rendering water radio-active has been invented by Dr. Hugo Lieber, of New York. His device consists in dissolving radium bromide in a suitable solvent, and then applying this to the inside of a celluloid cylinder and coating it with collodion for protection against loss. Tubes prepared in this manner and closed with rubber stoppers provided with stop-cocks permit the alpha radiations to pass into the calibre of the tube without restraint, and thus fill the cylinder with radium emanation. After allowing the emanation to collect for twenty-four hours, it is then a simple matter by rubber bulbs to force this emanation gently into a column of water. The details of this process were fully described by Dr. Lieber nearly ten years ago, and are now commercially available. In a similar way rods are coated with radium for surgical purposes. Water rendered radio-active in this manner loses this property very soon, and hence must be used as quickly as possible.†

Another valuable method of using radium is by mixing it with gelatine for use in injections and for applying it to the cavities of the body. This also has been devised and perfected by Dr. Lieber.

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* The agents are Morgenstern and Company, New York.

† Hugo Lieber, Ph.D., *Journ. of the Franklin Institute*, Philadelphia, December, 1911.

Radio-active water may also be made by placing pitch-blende in a jar of water, or suspending in it a coated tube or rod prepared in the manner above-mentioned.

From Bertier's observations radio-activity at Aix-les-Bains diminishes very rapidly from the moment of preparation of the bath. The mere fact of adding cold water and stirring the water of the bath is sufficient to lower the radio-activity of the thermal water 50 per cent.; subsequently the radio-activity is lowered more slowly. The following measurements are in millicuries :—

Radio-activity of the water as it emerges in tubs (temp. 40° C.)	...	...	0·62
,, ,, at 36° C.	...	...	0·31
,, ,, after ten minutes	...	...	0·25
,, ,, ,, twenty minutes	...	...	0·20
,, ,, in large pool (sulphur water).			

On allowing a current of thermal water to flow in the bath, the radio-activity is maintained at 0·34. In the pool, which has a large surface exposed, in spite of the large current of sulphur water which continues to flow in, the radio-activity is lowered to 0·157, the temperature remaining at 38·5° C. (101° F.). Consequently, to obtain the highest radio-activity, it is necessary to use the thermal waters on their emergence.

Hence, it is believed that the douche at Aix owes some of its virtues to the presence of radio-activity in the somewhat prolonged use, a prominent feature of the treatment at that Spa. The pools are used for the re-education of movements and the relief of traumatic ankyloses.

In ten minutes' use of the douche, Dr. Bertier estimates that about 500 litres of thermal water are used under variable pressures. The air is also saturated with radio-active emanation, and is inhaled by the patient, and this is held to be an important therapeutic aid. Bertier measured the radio-activity of this air contained in the berthollets, or small cabins, and finds it 0·16. He does not overlook the value of

the heat and humidity, however, as playing an important part in the cure. There is also found on the walls and in other places exposed to the waters a substance called barégine. It is composed of vegetable matter, and gives a peculiar feeling to the water, facilitating massage. This dries, when exposed to light and air, in the shape of flakes, and it also retains a certain amount of radio-activity, estimated at 0·09. The barégine can be collected in considerable quantity at the end of the bathing season in all the bath-rooms, but it is not employed therapeutically.

Returning now to the Yellowstone National Park, we find there about four thousand (4,000) thermal springs and about one hundred (100) geysers. Nowhere on the globe is such a marvellous group of mineral springs. One of the interesting features of Schlundt and Moore's studies was the discovery of thorium emanation in sixteen of the eighty-two gas sources examined. The thorium is associated with the radium emanation, and it is the first place on the American Continent where thorium emanation has been found. It was found in one instance to have an initial activity fifteen times greater than that due to the radium emanation in the same gas.*

Pitchblende yielding radium has been found in Gilpin County, Colorado, and carnotite, which yields uranium in considerable amount, is also found in that State and in Arizona.

It should be borne in mind that radium emanation is a gas with all the qualities of such a body; for this gas Sir William Ramsay proposed the name Niton. It decomposes under the emission of alpha rays, so that after three to eight days only half the original mass is left. The successive

* Herman Schlundt and R. B. Moore. "Radio-activity of the Thermal Waters of the Yellowstone National Park." *Bulletin* 395, United States Geological Survey, Washington, 1909.

Norris Geyser Basin. Radio-active Springs, Yellowstone National Park, U.S.A.
United States Geological Survey, Bulletin 395.

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decomposition products are termed Radium A, B, C, D, E and F, all of which are solid bodies in contrast with the gaseous mother substance. These bodies transform themselves with more or less rapidity, and, with the exception of B, also emit alpha, beta and gamma rays. Radium A, B and C have a half value of only a few minutes; D has a half value of forty years; E is of six days, and F of 143 days.

Professor Heinrich Kisch, of Marienbad, at the Sixth International Congress on Physiotherapy, held last month at Berlin, uttered a warning against the over-estimation of radio-activity as the potent factor in mineral springs. He made the very sensible remark that there is no reason to displace the well-known chemical and physical qualities by their radio-activity, or to hold that the degree of radio-activity of the springs runs parallel with their value. Ordinary spring water often contains more radio-active units than the well-known curative springs. Various saline and sulphated waters well known in balneology have only a slight radio-activity. Professor Kisch seems to hold that most of these natural radio-active waters hold an insufficient number of units to make them effective as radio-active waters *per se*. Even the emanatoria do not furnish sufficient units.*

At the same Congress Mache and Quess, of Vienna, and Ramsauer and Holthusen, of Heidelberg, reported on the absorption of radium emanation by human blood in treatment by inhalation and drinking. It seems possible to introduce the emanation into the blood and keep it there some time; how long is not stated. The possibility of exact dosage, according to the report, is in favour of the inhalation method; and the advantages of the drinking method are its great convenience and small cost.†

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* *Journ. Amer. Med. Assoc.*, May 24, 1913, vol. i, p. 1650.
† *Ibid.*, May 17, 1913, p. 1554.

A Radium Emanatorium is being installed in the Post-Graduate School and Hospital, New York, under the direction of Dr. Ludwig Kast, and, I believe, is now ready to receive patients. This hospital also has the apparatus for making drinking water radio-active, and so has the Hotel Chamberlin at Fortress Monroe, Va.

At the Johns Hopkins Hospital, Baltimore, it is proposed to equip an emanatorium. Dr. Howard A. Kelly of the hospital staff now has about $50,000 worth of radium, and will shortly have a supply of mesothorium. The advantage of mesothorium is partly in the fact that its life value is about eight years in comparison with the few days during which thorium or radium emanation can be used.

DISCUSSION.

Dr. A. JACOBI: I would like to know what the radioactive condition of all those wonderful mineral spas means for us. I am only a general practitioner and I want to know what they are good for. What do they claim the physical or theoretical effect is?

Dr. W. E. CASSELBERRY: I would like to inquire if the essayist has formulated an opinion as to the real efficacy of the radioactive qualities of the springs mentioned, or whether radioactivity is responsible for therapeutic effects long ascribed merely to the eliminative action on the skin by hot bathing and the conjoined regimen. For instance, Hot Springs of Arkansas; their chemical composition is almost identical with that of the waters of Lake Michigan, and it has been a mystery to what their superior effect might be due, other than to elimination, which seemed an inadequate explanation, because the same effect could not be produced from artificially heated Lake Michigan water. In a commercial way, radioactivity is alleged to be the powerful factor, and I am asking whether there is any ground to believe it to be the truth of the matter.

Dr. KAHLO (White Sulphur, West Virginia): I am glad Dr. Hinsdale has brought to our attention the subject of mineral waters and radioactivity, for, as has been stated by the previous speaker, the theory has been propounded that the difference in the effects of mineral water and the similar solutions artificially prepared is to be accounted for by the radioactivity of natural waters. As Dr. Hinsdale has said, this subject has been very extensively studied

in Europe. I have just returned from there, and took occasion while abroad to make some investigation of the same on my own account. Much importance is attached there to radium treatment in various forms, not only at mineral water resorts, where the waters are more or less radioactive, but also in the centres of medical teaching, where artificial solutions of radium salts are employed for this purpose. Most of you are doubtless familiar with the work that has been done in this field by Professor His, of Berlin, and Von Noorden and Falta, of Vienna. They have all used this treatment for different diseases, with varying results, but the results in many conditions have been unmistakably good. I think there can be no question as to the value of radium treatment in certain arthritides, notably in gout and rheumatism. How far the results obtained from the use of natural radioactive waters differ from those in which artificial radium salts are employed, I am not prepared to say, and, so far as I am aware, this question has not been fully investigated. The waters of Bad Gastein are, perhaps, the most highly radioactive of the well-known European spas, and yet almost equal importance is attached to this treatment at Carlsbad, Wiesbaden, and other places where the radioactivity of the waters is very much less. As you probably know, this treatment is employed not only by inhalation and by baths, but also hypodermically and by the mouth. For bath purposes it is usually prescribed in solutions of the same strength as the Gastein water. I believe this is a subject which will receive more and more attention from the members of the profession in this country, as we learn more of this property in relation to our own waters. Investigations of this kind have already been made in connection with a few springs, most of which have been found to be radioactive in some degree. Doubtless similar results will follow a further extension of such investigations. The technique in the use of the apparatus employed for this purpose—the fontactoscope—is not difficult to acquire, and but little allowance need be made for the personal equation in such investigation.

Dr. FREMONT SMITH: I beg to ask for information from Dr. Hinsdale whether there are any popular spas in Europe which are not radioactive. Also I should like to ask what is the grade of radioactivity of the springs of Hot Springs, Virginia, because that might be of some service in knowing where and when to send patients.

Dr. HINSDALE: Dr. Jacobi inquires about what value radioactivity would have for us in the treatment of disease. I have had no experience with treatment of patients by distinctly radioactive measures, although the Germans have published a great deal about that, and the work of Klemperer is probably the most complete exposition of it. They all seem to claim great benefit in treatment of rheumatism and gout and chronic affections of that type. We, I think, in this country have not received data enough to know

what waters are radioactive. The Government has not yet announced what waters of Virginia are radioactive. They have been tested by the Government experts, and I suppose we will hear more about that later, but I am in no position to make any publication about it. They are doing that work, however. As to whether there are any popular spas in Europe that are not radioactive, there is a very long list of European spas that are found to be radioactive, measured according to different scales as I have mentioned, and I think we will probably find that most all the springs of Europe are somewhere in the list. As to the proportion of benefit assigned to the radioactivity and to other features of the spa treatment, I may say that at Aix they do not claim that radioactivity is the principal means by which benefit is derived. They attribute there a great deal of benefit to the moisture and to the heat and to the manipulations which are given at Aix. Of course, they do assert that the radioactivity helps to some extent in the treatment of these cases. They are not dogmatic in their claims. I wish that I could give you more definite information about the radioactive springs of this country, but we are not in a position to make any definite statement about it; perhaps next year we may do so.

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Note.—My friend, Dr. Joseph H. Pratt, of Boston, has made an investigation of some of the "Radiogen" products with the aid of Mr. James H. Ellis, B.S., of the Massachusetts Institute of Technology. The samples tested were the Radiogenwasser and Radiogen earth. It was found that the water was 0·61 times as radio-active as an equal weight of uranium nitrate, while the earth was 2·22 times as radio-active, a considerably greater strength than most other radio-active materials, such as pitchblende. Mr. Ellis in his investigation determined the recovery curve of the activity of the samples. After boiling and driving off the emanation the radio-activity was nearly restored in from 200 to 300 hours, showing that the radio-activity is due to radium itself. The presence of radium in "Radiogenwasser" was conclusively ascertained and its permanence established. I am indebted to Dr. Pratt for an opportunity to add this note.

AN INQUIRY INTO THE CAUSE OF BRONCHIAL ASTHMA.

By ROBERT H. BABCOCK, M.D., LL.D.
CHICAGO.

In order to facilitate the discussion of the etiology of bronchial asthma the disease may be divided into two clinical forms. In one, the paroxysms of dyspnœa first make their appearance in early childhood and thenceforth occur with varying degrees of periodicity. They are induced by some irritant which the sufferer knows will excite his asthma, as, *e.g.*, animal emanations, odours of plants, some particular article of food, indiscretions in diet, &c. The paroxysms last a variable length of time and usually are arrested by some particular remedy, as the fumes of some asthma powder, a hypodermic of morphine, &c. During the asthma-free intervals the patient is free from dyspnœa and may feel entirely well. Lastly, the individual may be permanently relieved of his asthma by change of climate, especially when the exciting agent is found to be the pollen of some particular plant, although in some cases mere change of locality proves remedial even when no especial irritant can be identified.

These peculiarities have led clinicians to assume a neurosis as the underlying basis of the disorder, while the often prompt amelioration of symptoms afforded by some favourite remedy seemed to warrant the assumption of spasm of the bronchi, as well as the theory that the action of the

exciting agent was brought about reflexly either by stimulation of the circular bronchial muscles or through the vasomotor system and consequent hyperæmia and œdema of the bronchial and nasal mucosa. It is not necessary to consider other theories, such as inspiratory spasm of the diaphragm, Kurschmann's catarrhal bronchiolitis and the presence of Charcot-Leyden crystals, since these are but symptoms or manifestations of the underlying disorder.

Until within the last few years these manifestly inadequate attempts at explanation of the etiology of asthma were accepted and dominated our therapeutics. We were able to relieve the paroxysms, but not to effect a permanent cure; or, if by chance this latter did occur, we were not able to account satisfactorily for the fortunate result. But there has now come to the front an explanation of the phenomena of disease which rests not on hypothesis, but on demonstrated scientific facts, and which not only enables us to understand the symptomatology of bronchial asthma, but in many cases provides a prospect and rational method of treatment. This new conception of the nature of asthma, as many of you may know, is found in what has received the name "anaphylaxis."

Before elaborating this theory in its application to bronchial asthma, let me describe briefly the second form in which we recognize this distressing malady.

This type does not begin in early life, and there seems to be no inherited predisposition. It develops ordinarily after the individual has reached adult age and, according to my observation, very soon assumes the clinical features of chronic bronchitis with emphysema, but with paroxysmal exacerbations of the dyspnœa. The individual is distressed all the time, but frequently, perhaps daily or nightly, suffers from such an aggravation of his dyspnœa that it then truly merits the term, bronchial asthma. The disease is in

essence the same as the distinctly spasmodic or nervous form, but intervals of entire freedom from a sense of respiratory distress do not occur, or are abbreviated to a few hours it may be instead of days or weeks. Measures addressed to relief of the sufferer may be still efficient, but their effect is transient or less pronounced.

How now does the principle of anaphylaxis apply to these cases? In order to make this entirely intelligible let me give a concise statement of the essential facts of this enlightening view of the phenomena of disease.

By anaphylaxis is meant a protein sensitization or hypersensitiveness on the part of the animal to protein introduced into the system either by way of the alimentary canal or parenterally, that is, by some other channel which permits absorption. This foreign protein may be labile and active in the form of bacteria or protozoa, or it may be stable as egg albumin, and hence may be of animal or vegetable origin. So soon as this foreign protein enters the system of the animal, certain body-cells develop a proteolytic ferment whose specific function is to attack, split up and destroy, that is, digest the protein. In this cleavage process two groups of elements of the protein molecule are liberated, one a poisonous or primary group, which Vaughan [1] likens to the acid portion of a neutral salt, and another or secondary group which is not toxic. This secondary group, according to Vaughan, gives the distinctive characters to the protein molecule, and by him is likened to the basic element of a salt.

The splitting up, then, of the protein frees the poisonous portion of the molecule, and if this be in sufficiently large amounts produces toxic symptoms. The secondary and characteristic portion of the protein molecule also set free is that which sensitizes the animal to this particular protein. The next fact to be remembered is that the proteolytic ferment

generated by the body-cells is capable of destroying only the one kind of protein that called it into being and no other. For instance, if the protein be egg-white or a certain bacterium, the ferment can split up only the egg albumin or the particular germ and nothing else. Furthermore, this proteolytic ferment, which is developed for a definite and specific purpose, becomes stored up in the body-cells in the form of what Vaughan [2] designates zymogen or enzyme-producer, and in some instances the cells may undergo such profound changes that this zymogen may persist for a long time, possibly for years, and may even be transmitted from the sensitized mother to her offspring.

When, now, a foreign protein is attacked and destroyed by the specific proteolytic ferment of an animal's cells, that animal has become sensitized to that particular protein, but not to any other. If after a sufficient lapse of time, usually twelve or more days, a second dose of the same protein be introduced into the animal's body, the specific ferment at once attacks and splits up the protein with the production of the phenomena of anaphylaxis. If the protein poison be sufficiently abundant and the proteolytic ferment be capable of liberating a sufficiently large amount of the poison, fatal anaphylactic shock takes place. Otherwise the symptoms may be severe, but not fatal. Bacteria are themselves cells, and as such are able to generate a ferment which is capable of attacking and splitting up the protein of the body-cells. Hence if bacteria on entering the blood are capable of multiplying more rapidly than they can be attacked and digested by the fighting or proteolytic cells of the animal's body, symptoms of disease appear. But if, on the contrary, they can be destroyed before they can multiply to a dangerous number, then immunity is produced in that animal.

What now are the reasons for concluding that bronchial asthma is a manifestation of anaphylaxis? Of these the

most convincing are the observations of Auer and Lewis [3] in experiments on guinea-pigs. These investigators found that when these animals were sensitized to a protein, as, *e.g.*, horse serum, and then after a sufficient length of time were again injected with this protein, they at once manifested phenomena identical in all clinical features with spasmodic asthma, and died with extreme respiratory distress. On *post-mortem* examination the lungs were greatly distended, and the bronchi were so stenosed that air could not be forced through the contracted tubes. Furthermore, by suitable operative procedures it was proven that this extreme dyspnœa was not of central but of peripheral origin. In other words, the bronchial stenosis was due to peripheral stimulation or contraction of the circular or constrictor muscles of the air tubes. In short, anaphylactic shock in guinea-pigs is manifested by typical asthmatic seizure indistinguishable from the disease seen in man.

Additional arguments for the anaphylactic theory of bronchial asthma are found in the effects of the injection of horse serum on individuals who have suffered from asthma when in proximity to horses. Instances have been reported of severe and even fatal symptoms from the administration of diphtheria antitoxin to asthmatics, whose paroxysms had been evoked by exposure to the emanations from horses. Indeed, Rosenau and Anderson [4] warn explicitly against the danger of intense and even fatal anaphylactic shock from the administration of this remedy to persons sensitive to emanations from horses.

That such emanations are capable of producing anaphylaxis in sensitized individuals is found in the fact that such emanations contain sufficient protein to affect powerfully a previously sensitized person. This being the case, it is reasonable to conclude that the pollen of plants may contain enough toxalbumin to produce the same sort of symptoms.

All that is needed is a previous sensitization, and this may be acquired in early childhood, or it may be inherited.

The explanation of the cases in which an asthmatic paroxysm follows the taking of some special article of food or a hearty meal is not quite so easy, but yet can be found in anaphylaxis if we consider the chemistry of digestion. When food is taken into the stomach its various constituents are broken up by the digestive juices. The protein is attacked and split up by the digestive enzymes with the separation and liberation of the two elements of the molecule, that is, the poisonous and non-poisonous or secondary group, as classified by Vaughan. They are absorbed as amino-acids, and after absorption are reunited to form a protein that can be utilized by the animal for the construction of its own tissues. It is quite possible that owing to a relatively too great amount of protein in proportion to the digestive capability of the enzymes some of the protein is absorbed; then two things will happen, namely, the first dose of protein thus passing into the circulation will sensitize the animal to that protein, cheese or shellfish, or what not. Then, if after a sufficiently prolonged interval this same protein be eaten again, and again be absorbed, the phenomena of anaphylaxis will occur.

In this same manner any foreign protein that gets into the blood or lymph from whatever source in the animal body, the colon, pelvic organs, or some focus, as an infected gall-bladder or pus-tube, may be capable of inducing the symptoms of anaphylaxis in a sensitized individual.

This brings me at once to the consideration of those cases of asthma which develop years after birth and present no traceable hereditary predisposition. I refer particularly to cases with some disease of the upper air tract or the accessory nasal sinuses. Ever since Hack and others called attention to an etiological connection between rhinological abnormalities and bronchial asthma, patients have been subjected to

innumerable operations, sometimes with benefit, but more often without relief. My experience with this class of cases has not been extensive, and yet it has convinced me of the correctness of the view that naso-pharyngeal and sinus disease does play a most important *rôle* in the causation of asthma.

My observation is, therefore, in perfect accord with such reports as have been made, and quite recently by Justus Matthew from the Mayo [5] Clinic at Rochester, Minnesota.

Out of 184 cases of asthma operated upon, there were 157 instances of nasal disorders or of infection of the accessory sinuses, and of these cases 104 had reported themselves as more or less relieved, the degree of benefit being in proportion to the thoroughness of the operations. Inasmuch as all the 157 patients had purulent or sero-purulent secretions in the sinuses or pent up in the nasal passages, Matthew is of the opinion that the asthmatic symptoms were manifestations of anaphylaxis from absorption of bacterial protein.

Of my own cases let me cite briefly two or three. A lady physician seen last October had suffered from asthma in an aggravated form for a number of years. In accordance with my suggestion she consulted a nose and throat specialist, who discovered and drained an infected antrum. Relief from asthma was experienced for three months thereafter. She then had coryza with swelling of the middle turbinal and stoppage of the drainage from the sinus, and quite promptly her asthma returned.

A young woman who had suffered from asthma and hay fever since early childhood was found to have a double ethmoiditis and an infected antrum. Curettage of the ethmoid cells on one side and removal of the polypi were followed by partial relief. Circumstances prevented a more complete operation, and so Dr. E. P. Norcross decided to try the effect of mixed vaccines from a well-known

pharmaceutical house. Reaction was pronounced, but improvement began at once, and now this patient says she has her asthma only in so mild a form that she experiences only a feeling of stuffiness when the air is unusually damp or foggy. It may be added that the vaccines cleared up the antrum.

Dr. George Paul Marquis has narrated to me the case of a man sent to him from out of town with a double hyperplastic ethmoiditis. His asthma had been so bad for many years as to necessitate an annual change of climate during the winter months. Radical operation on his ethmoid cells completely cured his asthma.

Dr. C. was a sufferer from asthma for a number of years and was forced to spend his winters in Florida, to the great injury of his practice. Five years ago he discovered he had gall-stones, and last fall was induced to submit to their removal and drainage of the gall-bladder. The result on his asthma was surprising. His operation occurred last December, and he has written lately that for the first time for years he has been able to spend the winter at home and engage in practice.

Now how can we account for the occurrence of asthma in cases like the foregoing? The explanation is easy if we accept the theory of anaphylaxis. In chronic infection of the nasal accessory sinuses all the conditions are present, provided there is not free drainage.

At the beginning of the infective process the absorption of the foreign protein in the form of bacteria has sensitized the individual, and thereafter, when sufficient time has elapsed, each renewed absorption is shown by the phenomena of anaphylaxis and asthma.

In the course of time the symptoms of anaphylaxis become practically constant, and the sufferer is rarely free from his asthma, which torments him daily, or, as in one instance I have notes of, many times each day. With the

increased severity and frequency of the paroxysms, chronic bronchitis and emphysema become established, and then the asthmatic is never wholly free from dyspnœa.

It seems to me that this same explanation holds with regard to Dr. C., whose asthma appears to have been greatly ameliorated if not cured by drainage of his gall-bladder. Sensitization to foreign protein is just as possible in chronic infection of this viscus or of a pus-tube as in any other structure, and *apropos* of this latter condition may be cited the following case: A woman under the observation of my assistant, Dr. Bayard Holmes, jun., had suffered from hay fever for a number of years, but not from asthma. Some five or more years ago she got an infection of one Fallopian tube, and now has a chronic pyosalpynx. But, singularly enough, she states that with the infection of the tube she began to suffer from distinct paroxysms of asthma, and now her dyspnœa displays the features characteristic of the second form as I have designated it.

Nevertheless, although anaphylaxis appeals to me as the cause of asthma in chronic hyperplastic ethmoiditis and other sinus infections, we yet are confronted by the query, why does anaphylaxis manifest itself as asthma in one individual and not in another? In the present state of our knowledge we cannot answer unless we assume an underlying neurosis or inherited predisposition. Doubtless there are individual peculiarities just the same as in animals of different species. Why does the guinea-pig show anaphylaxis in the form of bronchial stenosis and extreme dyspnœa, while the dog, *e.g.*, shows pronounced disturbance of the gastro-intestinal tract? Hay fever, according to Vaughan, is a local anaphylaxis, but why does one hay fever sufferer have asthma in addition or develop asthma in the course of time, while another does not? As yet we cannot answer satisfactorily, and yet the theory of anaphylaxis is a long step in advance of the old

and purely conjectural hypotheses. Just here someone may ask, how can you explain the relief occasioned by remedies employed for that purpose? The relief from hypodermic injection of adrenalin is due probably to the increase in blood-pressure it occasions, since anaphylaxis is said to be attended with diminution of blood-pressure. But I cannot explain the *modus operandi* of morphin and various asthma powders unless in the same way as with whisky and ether. According to Vaughan, Besredka states that alcohol and ether prevent the phenomena of anaphylaxis for a time, and in this connection I might state that I once knew an old asthmatic who found relief from a stiff drink of whisky, reinforced by the smoking of strong tobacco, two remedies in which he never indulged except during his paroxysms. A patient of mine had been suffering from extreme dyspnœa and asthmatic exacerbations for four months when I first saw him. Suspecting some infection in the upper air tract, he was advised to see a nose and throat specialist. This he did, with the result that a pair of old cheesy tonsils were removed. Ether was the anæsthetic, and for three weeks thereafter he was entirely free from his asthma. Then he contracted what he called a "cold in the head," and promptly his asthma returned.

The practical application of the foregoing is this: in every case of asthma search for some focus of chronic infection in the nasal accessory sinuses, in a chronic hyperplastic ethmoiditis, or in some closed cavity in any other part of the body, and finding it, advise its removal by surgical interference. If the absorption of a foreign protein can be prevented in this manner, it is likely that the asthmatic seizures will be prevented or greatly ameliorated. Of course, in cases of asthma traceable to animal or vegetable emanations or to some article of food to which the person has become sensitized, we can do no more than advise avoidance

of exposure to the exciting cause. Nevertheless, the understanding of the etiology of this distressing malady certainly gives us a basis for rational and effective therapeutics in many instances that otherwise might go unrelieved.

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[2] *Idem.*

[3] *Journ. Exper. Med.*, 1910, vol. xii, p. 151.

[4] METZLER. See "Bronchial Asthma as a Phenomenon of Anaphylaxis." *Journ. Amer. Med. Assoc.*, September 17, 1910.

[5] *Journ. Amer. Med. Assoc.*, September 21, 1912.

DISCUSSION.

Dr. CASSELBERRY: We are indebted to Dr. Babcock for his concise and logical summary of our information on the connection between asthma and nasal disease in the light of anaphylaxis. Rhinologists have long been convinced of this relationship, but it has not been generally credited because its explanation was not lucid. Now, instead of ascribing to the polyps and congested nasal turbinals a reflex action, we know that they themselves are secondary to nasal accessory sinus suppuration which in turn occasions the asthma through toxæmia by absorption of pus. One reason why they cannot always be entirely cured is that, however skilfully or radically attempted, one cannot always completely cure the nasal accessory sinus suppuration; for there are ten or more sinuses, large and small, in each nostril, not being formed alike in any two persons, yet very difficult of complete access, since they border upon vital centres. This is especially true of the most distant, the spheno-post-ethmoid group; but having observed that the degree of benefit derivable from the surgical treatment of the nasal features of asthma is proportionate to the thoroughness of the operating, I have, of late years, supplemented antrum and fronto-ethmoidal operative treatments by subsequently removing whatever polypoid hyperplasia and pus may be found in the spheno-ethmoid sulcus and within the sphenoid sinus and post-ethmoid cells, with the effect, at the least, of a distinct amelioration in the asthma, and, at the most, by such a reduction in the number and severity of the paroxysms that not more than a residual asthmatic tendency would remain.

Dr. BROWNING: Briefly, some observations illustrating the minority cases to which the doctor referred, that is, to diseases coming within

the nasal passages. I recall one case where a patient had been operated on for chronic appendicitis and afterwards the asthma was much relieved. I have had two cases within the past three years of tuberculous glands, one in a child and the other in an adult, who were treated for the tuberculous condition by the administration of tuberculin, and the asthmatic condition has disappeared and so far has not returned.

Dr. C. W. RICHARDSON (Washington, D.C.): I have enjoyed the remarks that Dr. Babcock has made, especially in regard to the influence of the upper air tract, or anything that we do not understand of the asthmatic conditions. There is no doubt a great deal in this subject from the rhinological point of view, but nevertheless we cannot expect too much from this line of work. We who work in naso-pharyngeal diseases find very frequently that asthmatics are relieved, and very decidedly relieved, by treatment of the upper air tract, but it is often necessary to look further than the upper air tract in many of these cases. In a number of cases drainage will bring about relief, but in others we should look carefully into the general welfare of the patients, especially into the question of food. We will find that in three, six, ten or twelve months some will drift back into their asthmatic habit, and this must be remembered—this drifting back is sometimes due to the recurrence of the imperfect drainage of the nasal chambers. In some cases, unfortunately, this is not always so, and we could enumerate many in which the drainage of the sinuses and correction of deflections of the septum bring about permanent relief in these asthmatics. It is only necessary to cite one case that I saw some time ago which comes to my mind. The patient had been a severe asthmatic for several years, who came to me to have corrected a very badly deflected septum. The man was very tall and extremely thin. This was over eight years ago, long enough to satisfactorily explain the relief of the deformity as the cause of the cure of his asthma, on account of the fact that he has never had a recurrence of his asthma since the day he was laid on the operating table. When they come to me for intra-nasal operative work I always say I will relieve the intra-nasal condition, but cannot absolutely promise relief from the asthma.

Dr. BABCOCK: I do not claim that this explanation of the nature of bronchial asthma is going to enable us to cure every case of the disease, but it will give us a working hypothesis in each case. Thus the mere removal of nasal polypi is not sufficient, and the reason why this procedure has been followed by only partial or temporary relief lies in the failure of the operator to recognize or to remove the hyperplastic ethmoiditis responsible for the polypi. Then, again, we must remember that a person may become sensitized to more than one protein, as exemplified by the case of the woman who, though having hay fever, did not develop asthma until she got a pyosalpinx, since which time she has suffered from recurring attacks

of asthma. The well-known but transient relief procured by whisky was illustrated on the train coming to this city. There was on board a doctor who is a sufferer from asthma and who was not wholly free the first evening out. The next morning, however, he declared he was feeling absolutely well, and, as he said, because he had taken a Scotch highball at dinner the evening before. As stated in my paper, this is because alcohol is a temporary preventive of anaphylaxis. Taking all the facts together concerning this disease, I do not see how one can escape the conclusion that bronchial asthma is a manifestation of anaphylaxis.

THE RECOGNITION OF EARLY CHANGES IN THE LARYNX IN TUBERCULOSIS.

By W. E. CASSELBERRY, M.D.

CHICAGO.

Professor-Emeritus of Laryngology and Rhinology in North-Western University Medical School; Laryngologist to St. Luke's Hospital; Ex-President of the American Laryngological Association.

Not differing from tuberculosis elsewhere, the first definite change provoked by the bacillus in the larynx is an infiltration of the mucosa and submucosa by a diffusion of small, round, mononuclear cells, interspersed in which are collections of epithelioid and lymphoid cells, in the form of tubercles, which are deposited in large part in the connective tissue beneath the epithelial and structureless layers, hence, near enough to the surface to change its aspect in form and feature. Accordingly, the recognition of early changes in tuberculosis of the larynx will be promoted by keeping in mind :—

First, the disposition of tubercles and tuberculous nodules to assume a globular form which gives the surface of the infiltration, also termed hyperplasia, a mammillated aspect.

Secondly, their disposition to excite verrucous and granulomatous formations, which, though ordinarily delayed till

the stage of ulceration, may make an earlier appearance and give the surface a papillomatous aspect.

Thirdly, their disposition to excite serous exudation which may give the surface, not the proportions of gross œdema familiar at a later stage as "turban-shaped" and "pyriform," but an aspect of incipient œdema which, by a touch of pallid colour and somewhat tense consistency, imparts just the "atmosphere" necessary to lend distinction to the composition.

Moreover, although by no means exclusive, tuberculosis favours certain sites in the larynx, each of which tends to stamp the lesion, whether mammillated hyperplasia, verrucous formation, or incipient œdema, with its own local functional or anatomical mark, and, though the stamp be that of location rather than of lesion, its impress on a tuberculous matrix is apt to leave a mark distinctive of the lesion itself. Especially is this true at a site which, lacking a definite name, is herein designated, together with a description of its location and function, as the vocal angle (fig. 1).

Though a feature of the larynx, the vocal angle is not a separate anatomical part, but a line of junction of several parts, the terminals of which converge at an angle. In length the angle starts at the base of the vocal process, as represented by a small three-cornered area beneath the terminal ridge of the cord, and mounting with a postero-lateral trend, it marks the line at which the superficial structures of the true cord, the false cord and the inter-arytenoid fold merge into one. The function of the vocal angle is somewhat like that of a hinge, its overlying mucosa folding in and out with every movement to and fro of the vocal cord in speaking, and though under normal conditions it withstands unharmed this perpetual creasing, a tuberculous infiltration will cause it to retain, in the form of a furrow or fissure, the impress made upon it at the folding line.

The mammillated hyperplasia fundamental to the furrow is prone to make its first appearance, and the furrow to take its start at the lower extremity of the vocal angle in or near the small three-cornered whitish surface of the base of the vocal process. Although this is a point of greatest significance in the early recognition of tuberculous changes in

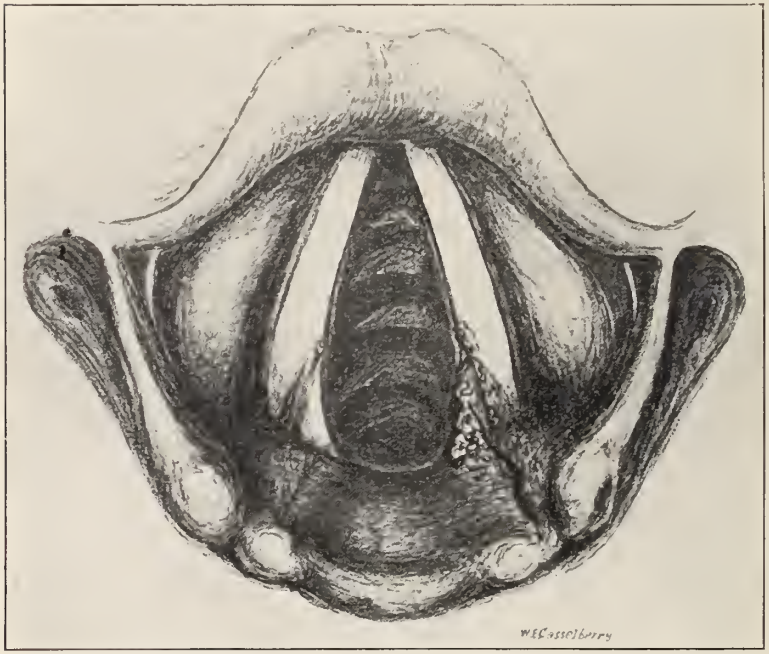

FIG. 1.—Mammillated tuberculous hyperplasia, with a furrow at the vocal angle, which is one of the earliest and most distinctive of the initial lesions.

the larynx, it doubtless is often overlooked because it lies beneath the ridge of the cord near its terminus, and only flashes into full view in the mirror during the moment of deep inspiration with widely separated cords, which naturally follows a prolonged vowel sound. In the half-opened position of the cords during ordinary breathing it is only

partly visible, and in the closed position not at all visible. The infiltration, however, is rarely limited to this one spot, and may make its first appearance anywhere along the vocal angle. Also, instead of at the base, it may first be perceived clustered around the apex of the vocal process, that salient point in the edge of the cord often erroneously referred to as if it were the whole vocal process, although in fact only its apex. The furrow in the beginning is simply the folding line or corner of the angle, given depth by thickening of its borders, but as the infiltration augments, especially near the terminus of the false cord, the furrow becomes more pronounced, and sooner or later it becomes a fissure, perhaps exciting granulomatous formation as it cuts into the rim of the larynx at the arytenoid.

Hyperplasia, therefore, of a mammillated or other typical aspect commencing at or near the subglottic portion of the base of the vocal process, and marked gradually by a furrow in the vocal angle, the author considers not only one of the earliest, but the most distinctive one of all the initial changes wrought by tuberculosis in the larynx (fig. 2). So soon may it appear that, in a semi-quiescent state, either alone or following interarytenoid hyperplasia, it may precede by months or years any more active developments; for it is now realized that, like its parent lesion in the lung, a tuberculous infiltration in the larynx may remain indefinitely in what is the equivalent of an early stage of development, perhaps in the end to flare up, or perhaps, as the author has elsewhere* shown, to recede, in the proportion, considering all types, of 20 per cent. And so distinctive is it that, by way of comparison, it may be said to indicate tuberculosis at a period before interarytenoid hyperplasia, if alone, will have passed the stage of similarity to non-tuberculous infiltrations. Even the broad-based centrally cleft, interarytenoid hyperplasia,

* W. E. Casselberry: *Journ. Amer. Med. Assoc.*, August, 7, 1909.

long regarded as characteristic of tuberculosis, before reaching its typical development, must pass through indistinguishable stages; for simple inflammatory hyperplasia having a crinkled, rugous and moderately elevated aspect, indistinguishable when limited to the interarytenoid fold from the early, tuberculous type, is not uncommon, especially in con-

FIG. 2.—Interarytenoid hyperplasia, which, if existing alone, is of little diagnostic significance at an early stage, as it is commonly simulated by non-tuberculous hyperplasia in the same situation.

nection with nasal accessory sinus suppuration, retention tonsilitis, asthmatic bronchitis, tobacco laryngitis, &c., and pulmonary patients, no less than others, are liable to these affections, so that not even in the presence of pulmonary tuberculosis itself will a moderate degree of interarytenoid hyperplasia, unsupported by infiltration at any other site,

advance the diagnosis beyond the question : Is it tuberculous laryngitis or simple laryngitis in a tuberculous subject? Excepting that any persistent hyperplasia in the larynx which supervenes in rapidly progressive pulmonary tuberculosis of the non-resistant type obviously requires no additional confirmation of its tuberculous nature.

It follows that the cases concerned in the problem of distinguishing between tuberculous laryngitis and simple laryngitis in a tuberculous subject are most of them of a hopefully resistant type, usually evidenced, despite a definite deposit in the lungs, by a fair state of nutrition, moderate pulse-rate, and but few bacilli, the interarytenoid hyperplasia in question being of moderate and slow development. Reference is made to this type by StClair Thomson as lupoid tuberculosis, but it is quite different, eventually running the course of laryngo-pulmonary tuberculosis and not that of lupus. The prognosis, however, is hopeful, and the actual results justify every effort at any sacrifice to invoke the methods most likely to secure its arrest; so that any loss of opportunity through lack of early recognition of that which later on is liable to become the major life threatening factor in the case, does an irreparable injustice to the sufferer. At the same time, any pronouncement of tuberculous laryngitis, based in error upon a non-tuberculous interarytenoid hyperplasia, is liable to work a corresponding injustice to him who, though a tuberculous subject, has but a simple chronic laryngitis; for the sacrifice of life's ambitions and business interests likely to be involved in the treatment and mode of life, under the idea of laryngeal tuberculosis, is far greater than in pulmonary tuberculosis alone. As a single instance may be mentioned the item of rest, which should include, not only rest for body and mind, but rest for the voice and rest from the exactions of business. If, however, an infiltration at the vocal process with a furrow in

the vocal angle be recognized, in addition to the questionable interarytenoid hyperplasia, the problem is then safely solved, the answer being laryngo-pulmonary tuberculosis (fig. 3).

It is somewhat later that the sign at the vocal angle reaches its most characteristic bilateral development, when,

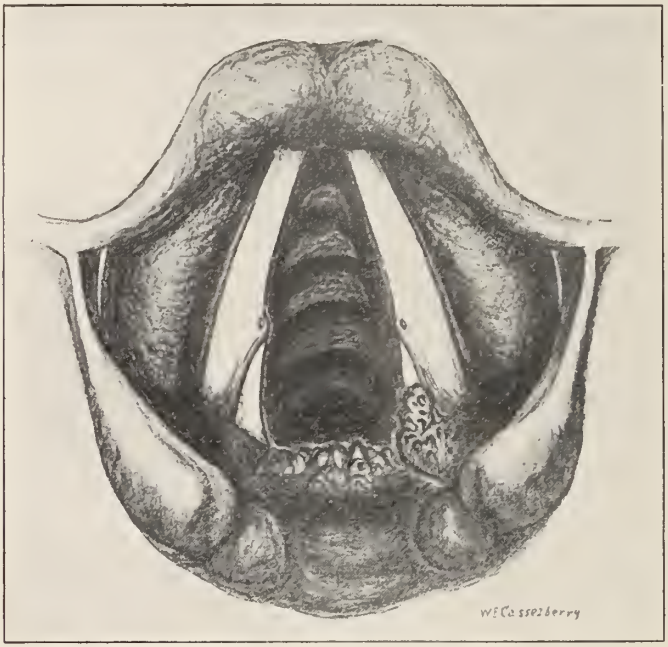

FIG. 3.—Interarytenoid tuberculous hyperplasia, which is given diagnostic significance by the accompanying hyperplasia and furrow at the vocal angle.

together with interarytenoid hyperplasia, it gives to the semicircular form of the posterior commissure a square or box-like effect, a veritable "squaring" of the semicircle. It forms a striking picture (fig. 4) believed to be sufficiently characteristic of tuberculosis of the larynx to justify a tentative diagnosis even in the supposed absence of pulmonary

disease which, on thorough search, will then be brought to light. That the prognosis still is hopeful is illustrated first in fig. 4, drawn in 1901 from the case of the Rev. J. W. D., in which the mammillated hyperplasia of the interarytenoid fold, and of both vocal angles with a furrow at the right and a fissure at the left, and with granulomata

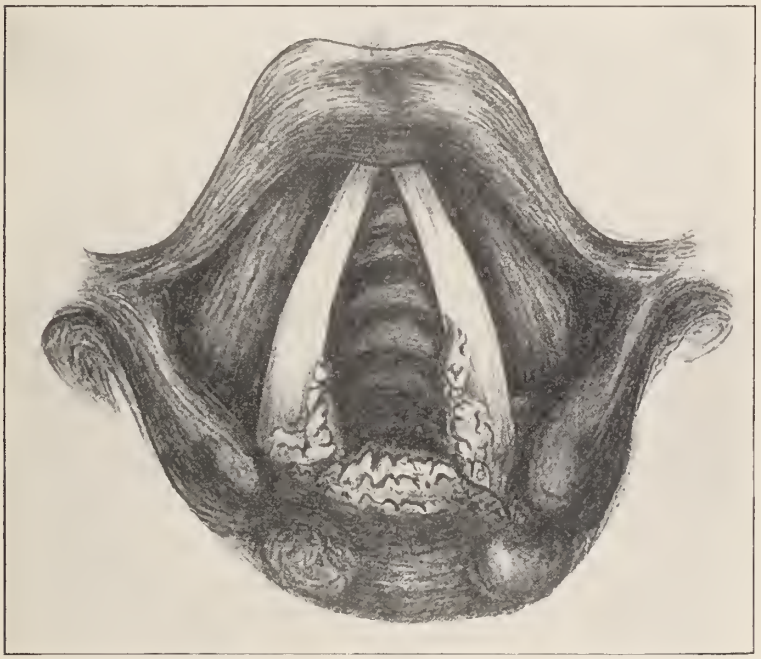

FIG. 4.—"Squaring of the Semicircle" in the case of Rev. J. W. D. The mammillated hyperplasia of the interarytenoid fold and of both vocal angles, with a fissure and granulomata in one and a furrow in the other, presents an aspect absolutely characteristic of tuberculosis of the larynx. Nevertheless, recovery ensued, as seen in fig. 5.

forming about the fissure, were absolutely characteristic of tuberculosis; and next in fig. 5, drawn seven years afterward from the same case, which shows that full recovery has ensued with respect to the larynx, a smooth scar, the

result in part of intralaryngeal surgical treatment, being the only reminder of its former state. The condition found to simulate most closely a tuberculous "squaring of the semicircle," is simple pachydermia laryngis, as shown in fig. 6, from the case of Mr. R. J. McR. It was differentiated by

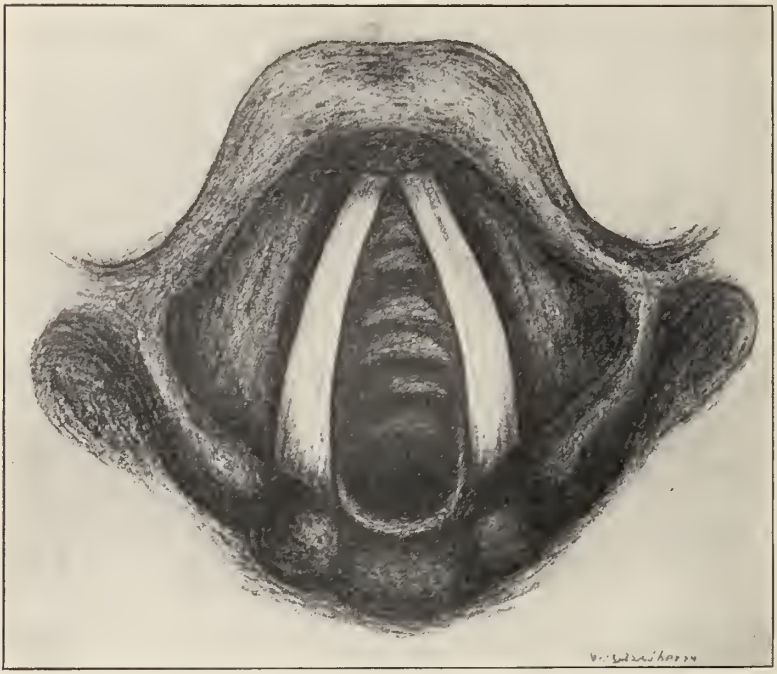

FIG. 5.—Case of Rev. J. W. D., seven years after fig. 4, showing the larynx fully recovered from tuberculosis, with only a smooth scar the result of treatment, which was in part by surgical measures.

the adjoining oval depressions and the absence of furrows at the vocal angles.

It is not to be inferred that the sign at the vocal angle has heretofore escaped observation, for although no mention is made of a furrow or fissure, it is discernible in various illustrations, including Nos. 3, 9 and 12 in the series of realistic

sketches by C. L. Minor,* who also describes as "very suggestive the early appearance of thickening at the posterior insertion of the cord, and a small white triangular ulcer," which he locates at evidently the same spot herein described

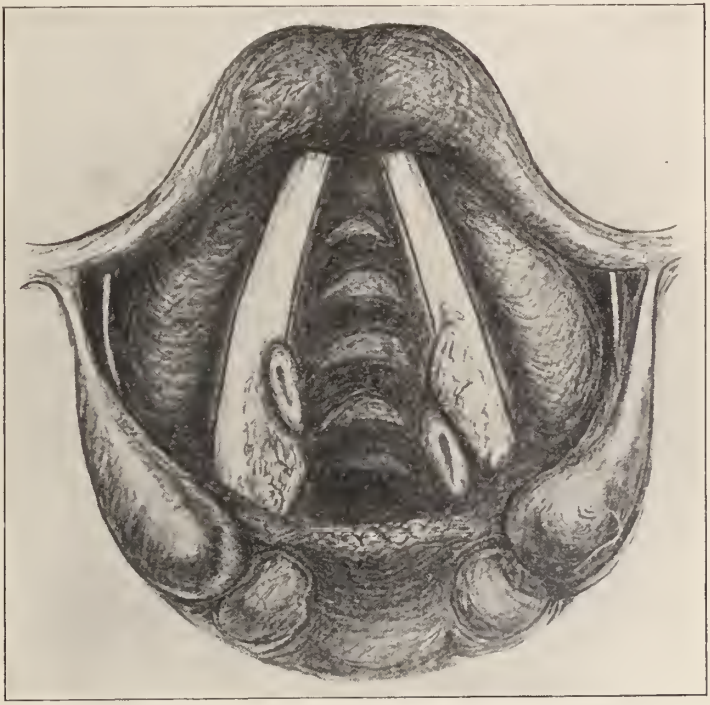

FIG. 6.—Non-tuberculous "pachydermia laryngis" in the case of Mr. R. J. McR. Differentiated from tuberculous hyperplasia by the adjoining oval depressions and the absence of furrows at the vocal angles.

as a three-cornered surface at the lower extremity of the vocal angle beneath the terminus of the cordal ridge.

The initial lesions at other sites, although not of frequent occurrence individually, are too numerous in the aggregate to permit of more than passing notice, limited to those which

* C. L. MINOR: *Journ. Amer. Med. Assoc.*, April 3, 1909.

may serve incidentally to accentuate the "motif" running through our theme; in effect, that the diagnosis of the earliest definite infiltration will be promoted, while keeping in mind the pathological trend, by an ability to recognize it, whether mammillated hyperplasia, verrucous formation, or incipient œdema, in the guise of the varying stamp placed upon it at different local sites. Take, for instance, lesions in the fibrous portion of the vocal cord, whose firm tissue bundles, by affording an extra lateral resistance, encourage the "fusiform" style of development, most characteristic of hyperplasia of the cord; the fusiform, then, if abbreviated and in multiple, becomes an "undulating or wave-like" infiltration of the cord; and if elongated and in duplicate it constitutes a type of "double cord." Again, verrucous formations and nodules in pairs, like simple warts and singer's nodes, seem to be led into these forms of development by vocal attrition of the cords.

The epiglottis, although rarely the first feature to manifest infection, provides the most favourable condition for exhibition to the naked eye of individual tubercles which, of course, represent in diminutive the elements of mammillated hyperplasia. Half the epiglottis of a case now in mind was marked by many greyish-yellow, pin-point, slightly raised granules scattered beneath a translucent, somewhat œdematous surface. Mention should not be omitted in connection with this unpromising initial condition that this patient made an excellent recovery with respect to the larynx, and a practical arrest with respect to the lungs, being now, fourteen years afterward, in an efficient state of health.

The arytenoid, exceptionally, may provide the first indication of a laryngeal complication by suddenly becoming œdematous, which, when pronounced, is an evil omen indicative of the non-resistant type, characterized with respect to the laryngeal involvement by speedy development, per-

sistent progress, and rapidly fatal termination. This is the type previously mentioned as obviously requiring no additional confirmation of the tuberculous nature of any infiltration which might occur in the larynx. Having stated in connection with its opposite, the hopeful variety, that the results of treatment justify any sacrifice, it is incumbent now to state that this non-resistant type should be carefully differentiated, no serious sacrifice in it being justified by the results of treatment; instead, these patients should be guarded from the privation and distress which surely follow in the wake of an indiscriminate exposure to the elements and to the hardships of travel in distant climes. Any attempt, likewise, at local surgery is but to court opprobrium and invite disaster, whereas, in the hopefully resistant type, with lesions not too diffused and reasonably accessible, intralaryngeal surgical measures have proven helpful in mitigating suffering, prolonging life, and promoting arrest.

Can a diagnosis, then, always be made from the laryngeal image alone? No, not always, for the infiltration may not have developed sufficiently, and it cannot be said that even a furrow at the vocal angle may not in rare instances be simulated by the hyperplasia of syphilis, pachydermia, &c., so that exceptionally every additional test must be applied in order to remove doubt or to make assurance doubly sure. Even then it must rest upon the laryngeal image to disclose that accentuation of the infiltration which constitutes a positive local reaction from a systemic tuberculin test, which, following the exclusion of syphilis by the Wassermann test, should dispel any remaining doubt.

DISCUSSION.

Dr. C. L. Minor: I am satisfied that there is too much neglect by our profession of the condition of the larynx in our tuberculous patients. A great many men examine the larynx as a part of the regular examination of the lungs, but too many good men still neglect it. Then I am satisfied that these slight changes which the doctor has referred to, need our close attention, for it is only in these conditions that you can do much with your therapeutics. Moreover, it is only the man who looks at all these cases carefully, laryngeally, who can make an early diagnosis of this trouble. Laryngeal tuberculosis certainly can be cured in quite a large number of cases if discovered early and rationally treated, but if neglected we know very well how little results can be had. Many men never look into a larynx until such a definite symptom as pain calls for attention, and then they are apt to refer the case to a laryngologist. Yet the report of the best laryngologist is not equal in instructive value to what a man has seen for himself. Since it is not difficult to master laryngeal technique, it is every physician's duty to be able to examine the throat of his own patients. You owe it to your patients to look out for these things. Dr. Casselberry has covered all the points so fully that it is scarcely necessary to dwell on them at all. However, I think we are apt to overlook in the epiglottis the slight thickenings, congestion of the upper border of which are easily forgotten, and yet which are valuable in diagnosis. Another lesion which I have frequently noticed is a moderate thickening of the false cords with slight pallor producing hoarseness by its pressure upon the true cord. These lesions, in my experience, are rather chronic in their course, but are unquestionably tuberculous in their nature. I cannot agree with the doctor in the slight importance he lays upon table-like elevations in the posterior commissure. So good an authority as Schrötter considers them pathognomonic of tuberculosis. Of course, the doctor sees so many non-tuberculous cases that he has an opportunity of judging whether the same condition can be caused by catarrh of the nose, but I believe that central elevations of this type are a pretty safe point in the diagnosis of tuberculosis of the larynx. I trust this excellent paper will impress upon us that a man has not examined a pulmonary patient satisfactorily until he has examined his nose and his larynx as well as his lungs.

Dr. Browning: I think that the importance of this subject is great. I feel as the President has just said that we have not completed the examination of the respiratory tract of a tuberculous patient until the nose and throat have been thoroughly examined. And there is another point which seems to me to be quite practical, and which should be considered in connection with the patient who is considered to be a suitable case for the use of tuberculin. I think that the larynx should be carefully watched, especially during the

beginning of its use. I think that it not infrequently happens that changes will be brought about in the larynx before they would be noticeable in the lung, before they would be noticeable in the temperature or other signs which we are accustomed to look for in the detection of tuberculin reactions. If the dose is repeated too frequently, or is too large, we may bring about disastrous results in the larynx, when by careful watching and graduating the doses by the condition of the larynx, we may bring about an excellent result. I believe there is a practical application of the principles which have been brought out here so well by the doctor which should not be lost sight of in the treatment of the tuberculous case with tuberculin. And also I was especially impressed with the suggestions on exposure of the patient to the elements. I am satisfied that the universal recommendation, without further consideration, that you should put all patients out of doors and unprotected, may have its exceptions. Not that the general principle does not hold good, but we must individualize in our cases.

Dr. KINGHORN: I would like to ask Dr. Casselberry as to treatment. I have often been doubtful whether to put the patient's voice at absolute rest suspecting a tuberculous condition, or whether simply at modified rest of the voice. I would like to ask whether he would put the patient at absolute rest.

Dr. CASSELBERRY: You may have noticed the effort in the paper to systematize the early lesions of tuberculosis of the larynx, and to formulate a rule of thought as an aid to their recognition while making examinations. The lesions are so diverse that I felt unless one could reduce their presentation, as well as recognition, to order, that little would be gained. Examination of the larynx is rendered comparatively easy by a preliminary 1 per cent. cocaine solution sprayed sparingly into the fauces, not mopped in with cotton, and it is harmless, if one tells them to spit out, and not to swallow immediately afterward. Furthermore, for those who wear glasses, the substitution for the ordinary head-mirror of a small, well-focussed, brilliant headlight, which eliminates one angle of reflection from the course of the light, has the advantage of disclosing small lesions in the larynx, with less blurring of outline. The direct method of laryngoscopy which is now fashionable I have found only exceptionally useful in this class of cases. Wih respect to the lesion of the epiglottis, the line of congestion mentioned by Dr. Minor, I included it in a way, but perhaps as representing a little later period. However, I think that you will find on close inspection of this line of congestion of the edge of the epiglottis that minute isolated tubercles are visible as the epiglottis is conditioned to show them best. The table-like elevation also mentioned by the Chairman I think is included in the paper. I agree that when typical a table-like infiltration of the interarytenoid fold is usually characteristic of tuberculosis, but I do not think it still an early lesion by the time it has reached this characteristic degree of development.

I am sorry Dr. Pottenger did not speak about the changes which constitute a local tuberculin reaction, as he has written so well on the subject, but you are all familiar with it. I referred to it as fully as I could; but we cannot always use tuberculin, having to make the diagnosis sometimes at least, off hand and quickly, and at other times there may be objections to the systemic use of tuberculin. I have tried in two cases to elicit a local tuberculin test reaction by local use only of a 1 per cent. tuberculin solution, swabbing the larynx rather vigorously with it, but as yet I have only negative evidence to offer, as neither case proved to be tuberculous. In reply regarding rest for the voice in treatment. Yes, provided it be tuberculosis of the larynx, and not merely laryngitis in a tuberculous subject. He should have perfect rest if he will take it. Absolute rest to the voice is one of the first principles of the treatment, but one must know that it is tuberculosis of the larynx before feeling justified in subjecting a patient to such an inconvenience.

THE RECIPROCAL RELATIONS OF THE CLINIC AND LABORATORY IN MEDICINE.

By T. W. HASTINGS, M.D.
NEW YORK CITY.

The clinic has been under obligations to the laboratory since the time of the first *post-mortem* descriptions of normal organs and of pathological changes in organs and tissues, and yet to-day one would pass over the founding of modern medicine by Rokitansky, Cohnheim and Virchow upon pathological and histological anatomy to the consideration of the more striking relations of the clinic to bacteriology, parasitology, physiology, physiological chemistry, and experimental therapy and laboratory diagnosis. It is not that pathological changes in tissues are unimportant, but in the newer fields of research there have developed so rapidly methods applicable clinically to the accurate diagnosis and to specific therapy and prophylaxis of infective and parasitic diseases, and the correction and regulation of toxic and antitoxic metabolic disorders of a non-invasive nature, that the earlier pathological investigations must be considered of a former era.

The laboratory has forgotten that the original stimulus for its efforts came from careful observers of clinical conditions. Without this it is doubtful whether the laboratory worker would have shown the patience of his clinical contemporaries, and it is likely he would have taken himself in despair to other fields than ministering to suffering humanity.

Within a few years the clinic has recalled this attitude of the laboratory as a fact, and with this recollection has swung far afield from the onward course of medicine, in the attempt to avoid the overwhelming strides of experiment and of laboratory application and from the practice of the results of experiment and observation, beyond the ken of the unaided eye and hand. It is interesting to note that the first scientific aid to the clinician was through the methods of Auenbruegger, Corvisart and Laennec, assisting the ear.

No one would decry the value of anamnesis, physical diagnosis, experiential and statistical knowledge, any more than deny the complementary relation of the laboratory to such methods; but in certain, if not in many instances, as diphtheria and epidemic diplococcus meningitis, statistical knowledge has made it evident that the laboratory has displaced the clinic in importance in the complementary relation. This is the fact most clinicians, the rank and file of practitioners, deny that there are diseases in which laboratory knowledge counts for everything, and that experience of thirty years develops less proficiency along certain lines than the modern first grade medical school imparts to its latest graduates.

I.—Indebtedness of the Modern Medical Clinic to the Medical Sciences.

To the fundamental sciences of physics and of chemistry more is owed indirectly than directly, excepting physiological chemistry, which has developed as a branch of chemistry essentially medical in application.

The chemical methods directly employed are of a simple nature and yet the chemical investigations which have enabled an understanding of normal and diseased processes have involved elaborate procedures on the part of highly

trained experts. This lack of applicability of advanced complicated methods is due to the fact that chemistry, *per se*, has to do in part with inorganic substances which have, so far as known, little to do with processes of disease and healing. The chemistry of organic substances, which play a considerable part in normal functions and pathological states, has been specialized in as physiological chemistry.

Apart from the manufacture of aniline dyes, non-physiological chemistry has more to do with the synthetic processes for the preparation of drugs than with the workings of a clinic.

From physics many methods have been selected. The stethoscope, the sphygmograph and sphygmomanometer, the various forms of cardiograph, instruments for hemometry, the spectroscope, the polariscope, the calorimeter, the Röntgen rays and radium emanations, to say nothing of the numerous principles of physics applied to instruments for the diagnosis and treatment of diseases of special parts and for mechano- and electro-therapeutics, are well known. The most widely used are the stethoscope, the sphygmograph, the Röntgen rays, the hemometric apparatus, and the polariscope. There was a day, not long since, when apparatus for electro-therapeutics was more widely used than far saner methods of therapy, but fortunately this day is passing. One still finds in the office of the country practitioner a bulky static machine, in place of even a few good medical periodicals, used for the treatment of all ills that are beyond the diagnostic acumen of the operator.

The application of certain principles of physics and the use of physical apparatus play a large part in modern chemical laboratory technique, but are subordinate, as a rule, to methods of physiological chemistry, biology, pathology and bacteriology. While for a time such methods were absorbed by the clinic, at the present time, along

certain lines, as in radiography, the clinic must employ an expert technician and an interpreter of radiographs. The progressive clinic must employ an assistant especially trained in this particular branch of physics.

To physiology, as to anatomy and general pathology, medicine in every branch owes not only its allegiance, but its existence, for without these three fundamental sciences inefficient empiricism would have prevailed, and no modern medicine would exist. It is true to-day that, with the exception of the application of the sphygmograph and the cardiograph for the pathological-physiological disturbances and the simpler physiological methods used in neurology, the methods of physiology are less applicable to the clinic than to experimental investigation. The highly developed apparatus for such methods must be used by one of special training in physiology.

From physiological chemistry many methods, simple and complicated, have been drawn, on account of their applicability as essential to the clinic for the daily routine for diagnosis. Some of the methods, as the testing of urine for albumin, sugar, indican, bile and blood, are available for any well-trained, proper-spirited physician, and for the fledgling hospital internes, who, through their laboratory training mainly, have proven a worthy asset to the clinic. Many of the methods, however, are too complicated and too time-exacting for utilization without the assistance of properly trained graduates in medicine, who have a liking for thorough study and investigation of cases.

This is no reason for not developing the facilities for conducting such work in connection with the clinic, and is an excellent reason for attaching to a medical clinic a trained laboratory assistant. In a well-conducted clinic, for example, the estimation of urea should be made in conjunction with the estimation of the total nitrogen and ammonia and other nitrogen containing substances in the urine. The

estimation, rather than the determination, of the presence of acetone bodies in the urine should be made by one qualified to do such work. For more advanced examinations for the investigation of problems in physiological chemistry in relation to medicine the physiological chemist must be not only consulted, but persuaded to become interested.

Experimental physiology, the elucidation of the nature of, and the treatment of certain thyroid diseases, and the understanding of circulatory and cardiac disease, stand out prominently among the achievements of modern medicine, as the result of infinite labour and patience of master minds persevering in the face of difficulties and discouragements.

The keen observations of certain contemporaries are comparable to those of Beaumont, Bernard, Gull and Addison. Since the prevalence of the diseases explained by these observations is relatively small compared to those investigated through pathology, bacteriology, and allied sciences, it is from the latter that the most beneficent results have come. To pathology and experimental pathology one must credit the understanding of the nature and progress of disease, and therefore the significance of a diagnosis and the rational employment of therapeutic measures directed particularly against the alleviation of symptoms, and the eradication of causative agents. Under the influence of pathology have developed bacteriology and parasitology, which have elucidated the ætiology of so many diseases, the nature of which pathology had previously determined.

As important as the discovery of the ætiological agents of infection and parasitic diseases has been the evolution of epidemiology, resulting in the elaboration of the methods for controlling epidemics of yellow fever, malaria, ankylostomiasis, trypanosomiasis, plague, and the gradual reduction of the incidence of typhoid fever. The discovery of "carriers" and the existence of such individuals dangerous

to a community, while not well recognized by the practitioner, and even doubted by some, should be impressed upon medical students as realities to be sought for under certain conditions and properly cared for. Recently attempts have been made in a few instances to determine the number of "carriers" among convalescent typhoid cases in hospitals and so regulate their relations with surroundings that they will not form a menace to the community. From experimental pathology and experimental medicine have come the effective methods of treatment for rabies, diphtheria, tetanus, epidemic meningitis, and the prophylaxis against variola. Whether from the clinician, the pathologist, the bacteriologist, such brilliant discoveries so-called have been the result of long, painstaking, scientific investigation by minds of the same type, utilizing laboratory methods. The clinician may be a keen observer and may recognize remarkable shades of difference in the symptom-complex of diseases, but the one who explains the meaning and causation of the symptom-complex must be the scientific investigator, exemplified by the leaders in medical science. Most recent are the brilliant results of scientific investigation in experimental therapeutics, and one hopes to see studies in chemo-therapy and in scientific investigation in materia medica place drug therapy upon a sounder and more wholesome basis.

From the simpler methods of examination, evolved through research in the medical sciences, have developed the clinical laboratory employing methods available for clinical diagnosis, and necessitating the training of men in cytology and bacteriology and microscopy.

The fact that clinical laboratory assistants may learn readily the simpler chemical and physical methods for diagnosis, but that it requires years of application to become familiar with, and expert in, clinical bacteriology, cytology, and the microscopy of parasitic disease, is not sufficiently recognized to-day.

II.—The Influence of the Laboratory on Modern Medical Education.

Experimental investigation in physiology, physiological chemistry, pathology, bacteriology, and therapy, have founded the conception, knowledge and treatment of disease upon a firm scientific basis and have made necessary the use of laboratory methods for diagnosis and therapy. Without experiment there would be little of science and accuracy in the practice of medicine.

The most striking change in medical curricula, due to the modern conception of disease, is the displacement of the didactic lecture and the theatrical clinic by demonstrative teaching, and the clinical clerk, or assistant-ship, system in hospital and dispensary wards. The student no longer learns the symptoms of disease by memory, and therapeutics by rote, but is made to see, feel, investigate and think before drawing a conclusion. The consultant of thirty years ago lectured to his students and conducted a private clinical laboratory with one or two assistants and followed with interest the autopsies on his cases, and to-day he teaches his students as assistants in hospital wards and dispensaries, and organizes clinical laboratories with capable assistants to whom he may send the students for a period sufficient to become familiar with the laboratory methods suitable for diagnosis. Students were thus formerly taught to diagnosticate through quizzing the patient and clinical observation, often superficial and perfunctory, since their minds were not stimulated to careful, minute observation by the habit of exactness, inculcated by familiarity with laboratory methods.

To-day a student is taught to diagnosticate through anamnesis, clinical observation, physical diagnosis, physiological methods, methods detecting structural alteration and disturbance of function in organs primarily involved, and

in other organs and systems secondarily disturbed; through searching for ætiological factors producing a general disturbance before function is altered in an organ or a system is attacked. Particularly important is the instruction given in prophylaxis and the prevention of the spread of infectious diseases. Experimental medicine and clinical laboratory methods have revolutionized diagnosis and to a great extent, though not so completely, therapy. The latter retains many of its deplorable features, not so much from faulty teaching as from the extended advertising and commercialism of drug-manufacturing firms, who, by individual canvassing, as well as by advertisement and distribution of pamphlets, attract the attention of the young physician as he settles down in an office to await the growth of a practice.

Away from medical centres and University surroundings the country practitioner should be more vulnerable to the advertised therapeutics than the city physician, but this does not always follow, since the tenor of present medical education leaves a habit or rejecting what is fundamentally wrong in practice. A deplorable attitude of the visiting physician and the practising physician is that of indifference to autopsies, both in endeavouring to obtain them and in checking up the clinical observations when the *post-mortem* examination is conducted by competent pathologists. The figures given by Cabot for the coincidence of anatomical diagnosis at autopsy with clinical diagnoses should impress anyone with the necessity for arousing interest in *post-mortem* examinations. Fifteen years ago the pathologists and a few progressive clinicians in this country had stirred some of the profession to activity, but of recent years, notably during the past five years, the clinicians' interest in gross pathology has diminished alarmingly. The complacency with which most clinical diagnoses are made, the self-satisfaction with which the therapy is conducted upon this diagnosis as a basis are evidences of a faith unbounded,

which should be replaced by careful and repeated observations to ascertain the facts before final judgment gives forth a diagnosis.

The figures quoted by Oertel, as those by Cabot, demonstrate how erroneous conclusions may be in medical practice even when based on painstaking clinical investigation.

This lack of interest in autopsies and this self-assurance in respect to clinical diagnoses have infested the hospital internes, excepting in a few institutions. It would be the better part of wisdom to insist that the hospital interne, if having through instinct and ambition no interest in autopsies, should be trained to appreciate their value, as the student is trained, and even made to perform, under the supervision of the pathologist, certain autopsies on cases from his wards.

The student in the best schools is trained to appreciate the knowledge gained through autopsies, but as hospital internes, under the influence of experienced clinicians, this appreciation wanes, and is often replaced by indifference to *post-mortem* examinations as to many other laboratory methods and other conceptions of disease taught previously by the laboratory departments.

Few clinicians are capable of performing autopsies, and it would not be desirable for them to undertake to perform them to any extent, for from the pathologist the most efficient reports are obtained, but the former, much to the satisfaction of the latter, might give evidence of a sincere interest in *post-mortem* reports.

The clinical clerk, or assistantship, system is a development *pari passu* with the laboratory and demonstration method of teaching. The lack of a sufficient budget, as a rule, limits clinical laboratory development in most teaching institutions, and the lack of realization of its importance limits it in hospitals, health resorts, and in private practice. Clinical laboratories will not be generally used until the

present generation of laboratory-trained students replace the passing generation of practitioners.

III.—The Practical Development of the Clinical Laboratory for the various Clinics of the Dispensary and Hospital; for Health Resorts of a Special and General Nature; for the Physician in Private Practice.

Under the preceding paragraphs laboratory methods applicable to the clinic have been mentioned.

The best system of utilizing such methods is under dispute, judging from the organization of laboratory facilities in different medical schools and hospitals. Whether each clinical department shall develop within itself laboratory facilities which it needs, or thinks it needs, according to its director; or whether a single clinical laboratory department to care for the needs, particularly along advanced lines, of the different clinics, shall be established, may depend upon many factors. In the first-grade institutions for teaching and practice there is no doubt but that the latter method is preferable as a system. The former method generally results in the assignment of the laboratory work to recently graduated assistants, who retain their positions, even as directors, for two or three years, so that the position is one of a rotating hospital interne or medical school assistant, which necessarily implies no familiarity with more advanced methods and therefore lack of efficiency in teaching. And, strange to say, this system still prevails in medical schools which first introduced the laboratory assistantship to the medical clinic in this country. This is not so on the Continent. It is wise to relieve the student of much of the advanced routine and to concentrate his attention upon some new or advanced method as an object of research.

Many advanced laboratory methods applicable to the

diagnosis of conditions other than those of the medical clinic should be made available, through a central clinical laboratory, for other departments of the school and hospital. In some hospitals the internes are pathological assistants during the first six months of service. The fault in this system is that after the sixth month the interne may do little or no laboratory work if so inclined and not interested, and since laboratory examinations are made for him, as for his visiting physician, he forms the habit of having his work done for him, and later on, in practice, of neglecting to have it done. Six months' training is not sufficient to form the habit of doing such work as may be done by the practitioner.

In other hospitals, and the number is few, a clinical laboratory well conducted by the director of the pathological laboratory or by a clinical pathologist is maintained, which results in the work being done on a high plane and in stimulating students and internes to interest themselves in laboratory study of cases throughout their service and thereafter in practice. This is by far the best system.

Again, in a few hospitals, the clinical laboratory work is conducted by young assistants attached to the medical and other clinics, and, under their supervision, by the internes, and while this method stimulates the internes and habituates them to the value of such work in practice, it maintains the laboratory service on a lower plane, since the recently graduated assistants, as directors of the laboratory and not under the supervision of a pathologist or clinical pathologist, do not reach the proper degree of efficiency in advanced methods during their short term of service.

The development of a clinical laboratory is, as a rule, considerably more advanced for medical than for surgical work and other clinics in hospitals. That this need not be so, and that a surgical clinic may benefit vastly by utilization of well-developed laboratory facilities, is evidenced by the superb organization at Rochester, Minnesota.

Laboratories established at health resorts, in this country particularly, are for routine efficiency as part of a money-making venture, and, therefore, even when well conducted in their beginning by able men, become obsolete in methods and fall below the standard through lack of facilities for conducting thorough experimental studies and the absence from the stimulating atmosphere of a University medical school or of experimental institutes.

The directors of health resort laboratories might keep in touch with, and even be proficient in methods if they had the habit of visiting occasionally University medical schools for courses of study, but, as it is to-day, such visits are for a few fleeting days to clear up a theory, or obtain superficial acquaintance with some one procedure demanded by their clientèle. There is no reason why such resorts should not take cognizance of the complications which afflict so many of their patrons as well as of the disease with a name for which they ply their therapeutics, and occasionally search for the causative factors in diseases with which they become familiar clinically. In the East, at Clifton Springs and at Saranac Lake and Liberty, one could find worthy examples to follow.

Within a few years there has developed in certain cities what may be termed a co-operative investigative association for diagnosis, the details of which may be obtained in Richmond, and in many smaller towns throughout New York State, and probably in many other States. Through a cordial co-operative system the laboratory knowledge of the recent recruits to the ranks of practitioners is made available to the mutual advantage of patients and the profession.

The influence of proper laboratory teaching is such that the student looks upon disease from a different point of view from that of one who lacks such training. This point of view is that of always searching for the ætiological dia-

gnosis, regardless of the name of the disease, and for the possibility of applying specific therapy, and results in the careful study of each case by all methods available. There is no question that the ultimate product, previously trained with such a point of view, is the more thorough diagnostician, and therefore the more capable of right practice, and this will be borne out by the next few generations of physicians who in practice will modify the present attitude of the average practitioner.

The length of time required for laboratory training is probably longer than for clinical training to bring one to the same degree of proficiency in each, and it must not be forgotten that during the laboratory training the student and instructor are in close touch with the clinical aspect of each case. The specialist is such through his familiarity with the clinical and laboratory methods of diagnosis and therapy, and the new generations of family physicians will come into their own again on account of their ability to apply laboratory methods to practice, provided the medical schools live up to their possibilities.

IV.—On the Advancement of Laboratory-trained Men to Clinical Positions.

If one recalls the progress of clinical medicine during the past thirty years one will appreciate that the most renowned and advanced clinics are those which have attached to themselves competent laboratory assistants and have been in close touch, amounting to co-operation, with the laboratories of the medical sciences or institutes of science allied to the medical sciences, and whose directors are men well trained in laboratories, as in Baltimore and Munich.

As feared by some, there is danger in giving clinical positions to men who have done purely experimental work,

and little of it, with one or two experimental successes, and to men long trained in one medical science. A man of such training with no clinical experience must of necessity submit to clinical training before efficiency for holding a clinical position is obtained, even as a clinician who has not previously had the requisite laboratory training, which is considerably more than there is time for in a medical school curriculum, is unfitted to direct a progressive medical clinic.

One cannot agree with many of the recent statements of Bevan.* Laboratory-trained men are not against the clinician, but would have the clinician made after a new pattern, thoroughly learned in laboratory methods through three or four years' experience while a clinical assistant, so that he may understand and interpret properly the utilization of physiological, pathological, bacteriological, and biochemical methods for diagnosis and for therapy.

Professors in medical sciences should be men who know considerably more of the subject they teach than the clinically trained men in medical schools ever hope to know, on account of the little time the active clinical man can give to keeping pace with any medical science. The latter are not suitable for medical sciences, since the facts in detail, the truth entire, should be known by one in order to teach the sciences, and it is impossible, with the tremendous development in the past twenty years, to be trained to teach in the medical sciences unless a specialist, and often a specialist in one branch of a medical science.

Those of whom Bevan speaks as capable of holding advanced positions after passing from the medical school and through their hospital experience to specialization in some branch of medical science, should be the assistants in clinical work and eligible for advancement.

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* Bevan: "Medical Training and the Hospital," *Journ. Amer. Med. Assoc.*, 1913, vol. lx, p. 974.

THE FRUITS OF LABORATORY WORK IN RELATION TO CLINICAL TUBERCULOSIS.

By DR. EDWARD R. BALDWIN.

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The habit of making distinctions between clinicians and laboratory workers has grown up in latter times. It was inevitable when the increasing complexity of medical science made a subdivision of labour imperative. The result has been unfortunate in some respects. Inability of the pure clinician to appreciate the laboratory specialist, and, on the other hand, a contempt for the art of medicine—at least, the empirical side of it—on the part of the laboratory man has ensued.

They should be brought together, as there is no such line of demarcation between them as some would establish. I believe it is indolence or love of good dinners that keeps some men from laboratory work, not lack of aptitude. Or they are so tired out in making the day's rounds that they have no stomach for it. Well, then, they must needs become one-sided, and employ someone else to do what is now indispensable many times for diagnosis and treatment.

When you ask what fruits have come from laboratory work in tuberculosis, one naturally harks back to Koch's discovery of the bacillus, but really one should go still further back. Villemin, in 1865, properly began to bear

fruit, and since Koch we have never ceased to improve our diagnosis and treatment with the aid of laboratory studies. It is true much wasted time has been given to it, but so it is with the physician at the bedside. False deductions in the laboratory, as well as by the bedside, have cost many lives. This is the way medicine blunders along toward an exact science, but never reaches it.

Probably no single fruit ever equalled the discovery of the bacillus in importance; a close second was tuberculin, the value of which is now being appreciated As an addition to treatment tuberculin has not been such a boon, but in revealing the enormous number of infected individuals, both human and bovine, the whole programme of tuberculosis prevention is influenced. The ideas of inheritance were so dominant in the past, it is painful to think what would have happened but for Koch's work. The revelations from it are painful enough anyhow when we think what widespread fear of infection has done for the tuberculous victims. Nevertheless, let us consider what the laboratory has done directly or indirectly for the diagnosis of tuberculosis in addition to the bacillus.

Tuberculin is occasionally of great value in excluding tuberculosis when no reaction is elicited. The blood-culture differentiates between typhoid and miliary tuberculosis in many instances. The bacillus tuberculosis may even be found in the blood at times. By inoculations of guinea-pigs, not only the blood, but the urine may reveal with certainty the nature of the malady. Without this test of the urine, diagnosis of kidney tuberculosis in an early stage would be very difficult. Likewise, cultures of the blood, sputum, and urine may give information of value as to what other infections may complicate tuberculosis, thereby furnishing the material at the same time for a possibly successful therapy, otherwise wanting. A skilful clinician may dia-

gnose malaria, sarcoma, and leukemia without a blood examination, but rarely with that certainty which accompanies the use of the microscope. A leukocytosis from a concealed suppuration, an appendical abscess, or a pyelonephritis may be found important to know for diagnosis, and this was also the result of laboratory researches. It is so customary to turn to the microscope for guidance nowadays, that few of us realize how much more accurate diagnosis and treatment have been made by it. The misfortune is that so many are still content to leave these methods for experts or consultants and not perfect themselves in their use, at least enough to understand them.

While on the subject of differential diagnosis, I ought to mention the use of lumbar puncture, of the examination of exudates aspirated from joints, the pleural and peritoneal cavities, as well as from abscesses. There is also the differential diagnosis by examination of excised tissues where cancer, syphilis, and tuberculosis are to be considered. Less can be claimed for the agglutination test for tuberculosis than for typhoid fever, or for the complement fixation test by means of the serum. It must be confessed that they are too uncertain to have anything but scientific interest. The opsonic test falls in the same category of the impracticable. Nevertheless, it may be remembered that certain principles have been worked out by their aid which are valuable in treatment. I refer, of course, to vaccine therapy of various kinds, with which we should class tuberculin and so-called auto-inoculation. A knowledge of the effects on the blood, on the local lesion, on the secretions and excretions of the administration of tuberculin, or of the absorption of auto-tuberculin from the disease foci, cannot fail to make the phthisio-therapist better equipped to manage his patient than the man who knows no more than what outward signs tell him of their meaning. It is true that much

that has been learned empirically about the treatment has simply been confirmed by laboratory study, but it has given us more delicate indications, more definite rules and means of control. Foolish or injurious therapy has been discarded in consequence of tests made in the laboratory. Theories have been born and have died in the laboratory; it is well to acknowledge it. On the other hand, what a blessing that they were not all tried on the patients entrusted to our care! Too many bedside ideas have been born of criminal ignorance that tuberculosis is a deep-seated infection that cannot be reached by medicaments as harmful to the tissues as to the bacillus; many such things have faded out of memory, but some, thanks to the laboratory studies, have been hastened into oblivion. Unfortunately, nowadays it has been our misfortune to see many new remedies or systems of treatment exploited in the name of science—I mean pseudo-science, and false claims made for them on the basis of false experiments, either ignorantly or dishonestly performed. This phase of the laboratory is certainly the present-day curse, and no relief is in sight except from a vigorous Governmental control.

Looking away from the immediate application of laboratory results to the patient, let us survey the widespread influence research is having on society, on national, State, and municipal health laws, on the control of bovine as well as human tuberculosis. The direction of these agencies, their methods, and their success depends largely upon the painstaking studies on infection, the ways of transmission of the bacillus, the best methods of the disposal of sputum, disinfection of rooms, personal hygiene and treatment, to a large extent deduced from experiments.

Nor do I think we have everything settled by this means. Many things have to do with human conditions that the "try it on the guinea-pig" rule will not avail to guide us. It is

for principles chiefly that we are to look to the laboratory; their application requires the wisdom and common sense of the practical man of affairs, but let him not ignore the standards set by scientific knowledge. The public has been led to expect all the ills of human flesh to be cured by some serum evolved out of a laboratory. Let us do our best to give them a saner view than this without discrediting the real good that comes from it. The fruits of the scientific laboratory may mitigate and possibly arrest the ravages of a disease like tuberculosis, but the victim must needs reap his crop from a broken constitution or his evil habits. If this truth were better understood, the disappointed ones would less often jeer at our failures. The laboratory needs defence for its shortcomings just as does clinical medicine, but it needs more support from the public. Progress in medicine is dependent upon more hearty co-operation between the man at the bedside and those at the microscope. The former has nearly all the advantage of direct contact with the patient, and an intimate acquaintanceship which develops confidence. Let him generously admit in his intimate talks to his patients his indebtedness to the laboratory for valued help in diagnosis and treatment. Thus will the best fruits in medicine be brought to ripening.

DISCUSSION.

Dr. CASSELBERRY: I must say it never occurred to me that the laboratory is in need of defence; instead, those who fail to employ its advantages may well feel in need of defence. I think its justification can be summed up in two words—antitoxin and Wassermann.

Dr. MINOR: I felt that this subject should be brought up for discussion here, as it is one, these days, which is of very vital importance. We all realize that no army can get along well unless the various members of it co-operate, the commissary is as essential as the general staff; but it seems to me that we must remember that the commissary is only the commissary, and must not try to be the general staff. We all recognize and respect the laboratory worker, and we

realize fully that without his aid we are helpless to make advances that we need to make in these days, but as Dr. Baldwin realizes, it is impossible to-day for a man to be a laboratory worker and a clinical worker in a large practice. The demands of the laboratory upon his time are insistent and have to be met, the demands of the patient are insistent and have to be met, and the two cannot, to any great degree, be combined. My complaint is, and it is a very real one, that for reasons which cannot be easily explained, there is a distinct arrogance in the attitude of the modern laboratory man towards the clinical worker. He unquestionably has done great things. It was the result of the laboratory work which gave us diphtheria antitoxin, and a great many modern remedies of great value, and I would not belittle what the laboratory has done, but I cannot admit their claim to the exalted position they would arrogate to themselves. However important they are, they must come second to the clinician. We all admit that the plain clinical worker has done in the past, and can still do for sick people, splendid work without the assistance of the laboratory man, while the laboratory man is perfectly unable to do anything without the clinician; in fact, his work is pointless unless it is to be applied finally in the clinic. Therefore, he must necessarily be secondary to the clinical worker. Now, my contention is, that he thinks that his position is not secondary, but primary, and I believe most men will agree with me that they are showing such an arrogance as I have referred to. Not, it is true, my freind Dr. Baldwin, but the average laboratory man to-day does show this mental attitude. This was why I asked such a typical laboratory worker as Dr. Baldwin, who yet combines the clinical and laboratory methods in a happy way, to prepare this paper. I feel that in the long run the balance will be restored, and the clinical worker but show the way to the laboratory man in his work. The final effort in medicine is therapeutical, and medicine which does not result in therapeutics is useless, and the world would reject medicine to-morrow, however brilliant its studies, unless these resulted in the good of the patient. Now, we have to-day too many laboratory workers who never think of anything but the laboratory side, who take too little interest in its practical application, and who look down upon the bedside worker. To get the best results, the laboratory man and the clinician must work hand in hand, each respecting and valuing the work of the other, and both realizing that the patient's good is their final aim.

Dr. A. K. Stone (Boston): I have been in the habit of saying to my students that there is no laboratory procedure that cannot be called into question. Even the almost indisputable bacillus of tuberculosis may be disputed, for there are other acid-fast bacilli that come in at times.

The laboratory man must be kept as an assistant; he is the helper, not the superior. I think that is very important to keep this constantly in mind. It seems to me there is nothing that gives a man

more pleasure than to have enough laboratory knowledge to be able to go in and check up laboratory findings for one's self; and even if a man cannot go ahead and make new examinations and new discoveries, he can have lots of fun in the laboratory, and it is well worth every man's trying to take a little time off for this purpose.

Dr. F. M. POTTENGER: Following further in the line of thought of the previous speaker, the reason why a good clinician, as a rule, cannot do efficient laboratory work is because these two lines of work are adapted to an entirely different type of mind. The clinician is essentially an optimist, and he should be. He is a man who goes along and does things to obtain results. He sees the needs of his patient and furnishes them. The laboratory man, on the other hand, is slow, patient, and ultra-critical in his work. The clinician who has worked hard all day, seeing his patients and carrying in mind their ills, cannot sit down and patiently work out a laboratory problem. I have observed that most men who undertake to do both clinical and laboratory work, as a rule, neglect one at the expense of the other. One other factor regarding laboratory work. The laboratory man is apt to believe that his work is exact. It is not. We clinicians recognize that our clinical work is not exact; at the same time it is often as exact as that of the laboratory man. We know that if we are clinically correct in 60 or 75 per cent. of our diagnoses we are doing well. Most of us fall far from that. The laboratory man feels, however, that when he makes his blood count, or when he examines the urine or sputum that his result is accurate. Nothing could be further from the truth. Slipshod methods in the laboratory are just as often found, and will lead to just as much error as they will in clinical work. Now, if we will recognize these facts and learn that the laboratory is our greatest helpmate, if we use it right, and also that it is not as exact as we have been thinking, then, and not until then, will the laboratory and the clinical man be on the proper basis.

Dr. KINGHORN: I would like to say a few remarks on what Dr. Pottenger and yourself have said. First of all, with regard to Dr. Pottenger. He said that the two could not work together; that is to say, a man cannot work in the laboratory and in clinical medicine. Now the physician that I recognize to-day as being the first physician in the land, for what he had done both clinically and in the laboratory, belongs to this Society, Dr. Trudeau. As you know, he is a great clinician. There is no one, I think, who can equal him in his clinical knowledge of tuberculosis. No one else can question the rank that he has attained as a laboratory worker in tuberculosis. In other words, he has combined in himself a great clinician and a great laboratory worker. I can also refer to his first assistant, the Vice-Dean of our School up in Saranac Lake, whom I have often thought of as the Elisha, and who is the writer of this paper; and Dr. Baldwin certainly has combined those two qualities. He is a clinician of world-wide renown; and he is also a laboratory worker of world-wide renown, so

that I think this shows that these two qualities can be combined in one man. Now, from my observation of the trend of medicine in late years, the physician has combined clinical study with laboratory study. For instance, the Rockefeller Institute was started primarily for investigation. What have they done? They have their hospital right alongside their institute, so that the two can be combined right there. The whole trend of medicine to-day, I think, is to combine the two. Dr. Minor has said that a man with a large practice cannot do both. I say that in order to meet that condition do not let a man have such a large practice. There are other men that are coming along that need the help, and let them share the practice. I am sure, because I have combined laboratory work with clinical work; and I know that you get great principles of treatment from the laboratory that you would not get by reading, and that you will only get them by the laboratory, and I say the whole trend of medicine to-day is to combine clinical and laboratory study.

Dr. F. M. POTTENGER, replying to Dr. Kinghorn: I still maintain that these are the exceptions and not the rule. I think we all recognize that Drs. Trudeau, Baldwin, and Kinghorn are exceptions in their work, and that they have been able to do exceptionally good clinical and laboratory work, but that does not prove that it can be done generally. I cannot do it.

Dr. BALDWIN: I do not think there is very much to say. You all agree with me that the well-rounded man has to know something about laboratory work, although he cannot do very much. As for Saranac Lake, this mutual admiration society which Dr. Kinghorn has started up leaves me no answer! In regard to the man who is doing so much practice that he cannot keep up with the laboratory work, I think Dr. Kinghorn's contention is correct. No man should feel himself so desirable to his patients that he cannot take help. He can turn his patients over to somebody else, and the only way that you can comprehend the field of medicine—and I may say the field of tuberculosis to-day—is to have plenty of help, and I mean by that, help in the practice of medicine; and if you are going to keep up even with the new things that are coming up all the time, a man has to be well read. Then another thing: a little laboratory experience makes a man more self-critical. I do not say it makes him any better practitioner; I think sometimes it interferes with his being a good practitioner, simply because he does not give his patients so much hocus-pocus—so much talk!—but after all, the patients get more confidence if they live long enough with him. A man who is doing both ought to be theoretically better rounded. I have had plenty of experience in both ways, and I realize my deficiencies in the laboratory work, and I also realize the tremendous advantage that it has given me, and I speak of it modestly because I realize so many mistakes that I have made in clinical work too. Do not expect such accuracy in the laboratory that you need to consider that as the final word.

THE RÔLE OF PHYSICAL EXERCISE IN THE OPEN AIR IN THE PROPHYLAXIS OF TUBERCULOSIS.

By JAMES M. ANDERS, M.D., LL.D.
PHILADELPHIA.

Systematic physical exercise, as a means of prevention in, and the ultimate cure of, pulmonary tuberculosis has not received that degree of recognition at the hands of the medical profession that its merits warrant. Unquestionably, there has been a disposition on the part of physicians to overlook the signal importance of muscular exercise, since the general acceptance of the principle of rest in the treatment of all cases of this disease in which fever is present. My object is not to attempt to underrate the great value of rest as a remedial agent in the active stages of pulmonary tuberculosis, but rather to emphasize the first-rate importance of good muscular development as a means of minimizing the number of susceptible persons.

Efforts to prevent the occurrence of disease were developed to a high standard long before the discovery, through bacteriological research, of the pathogenic organisms of disease, especially by the Greeks and Romans. Later epochs, however, seem to have fallen into an opposite tendency. Among the recognized agencies to keep the body in health and to prevent its germicidal power from running low, an adequate amount of muscular activity is of the utmost

importance—more than that, its general adoption would have a vital effect upon the far-reaching future of the American race.

As members of the medical profession, our interest in the national physique and the evenly balanced condition of mind and body of the American race, not only during school life, but also during later life, should be greater than that of any other class of society. The question may well be asked in all seriousness: whether the medical profession has lived up to its reasonable responsibilities with regard to the tendencies to push the head at the expense of the muscles and sinews, at school and universities, and to retire from business careers physical bankrupts at or before fifty years of age?

It is an indisputable fact that the American race, as a whole, is slowly undergoing muscular deterioration, of which one of the principal causes is insufficient physical exercise out of doors, or, in other words, our mode of living as a nation. Two additional considerations to be reckoned with in accounting for our lack of physical stamina are: the ingestion of physically inferior races from abroad, and the undoubted trend away from occupations requiring the use of the voluntary muscles. It is difficult to understand the lack of interest by the medical profession in the opportunity opened by this state of affairs to point out to educators the supreme importance of keeping the body strong and healthy as the prime requisite in racial supremacy.

It is an accepted principle that increased respiratory exchange, the result of muscular activity, improves the metabolic processes and general health of the body. It must be recollected, therefore, that the endurance or resistance of an individual is in direct relation to his muscle power of muscular development, which, it has been well said, means his "physical development."

Of special interest to this Association is the fact that climate also exercises an influence of some moment upon the standard of physical efficiency. For example, in general terms the muscular development of people residing in cold or temperate climates is better than that of those residing in warm climates. True, the latter take less bodily exercise, "but this is off-set by taking less food, augmented skin action, and, therefore, increased circulation." Moreover, the physical development of "people living in the interior is better than that of those living on the coast, and in the mountains better than in the interior. People in the interior are usually farmers whose occupation necessitates an outdoor life and the use of the muscles; the reason for the hardiness of the people in the mountains is, of course, apparent."

The struggle for prevention in tuberculosis and other infective diseases can be, in the present state of our knowledge, carried on both intelligently and effectively by directing efforts primarily to the specific cause, including the prevention of infection, and by the removal of favouring conditions and predisposing causes connected with the individual. In the case of pulmonary tuberculosis, the specific cause rarely becomes operative unless predisposition exists.

From the standpoint of both prevention and cure in pulmonary tuberculosis, our most effective means are those that enable the human organism to resist the infection. We cannot annul the tubercle bacillus by a direct attack, although there are known means to prevent exposure to infection, and these are to be advised and encouraged. Unless it be found practicable to permanently immunize human beings by the inoculation of non-virulent living tubercle bacilli, then efforts to raise the standard of physical efficiency to the point of establishing natural immunity may not be relaxed.

Movements to prevent tuberculosis must also include the

isolation and institutional treatment everywhere of advanced tuberculosis cases. The departments of health officials should be educational forces in the community, instructing the people among other things in the principles and details of the prevention of tuberculosis. Educational work of this character should begin among the children of both sexes in our public schools.

In dealing with the problem of the prevention of disease the individual is regarded as the unit of a mass, but in the case of tuberculosis we can deal with human beings as individuals, and by selecting persons markedly predisposed to this dread disease, enable them to bring about a condition of immunity, as the result of systematic physical exercise, and other measures. Obviously, the prevention of tuberculosis involves something more than physical exercise persistently carried out, but its commanding position in accomplishing a state of physical development that successfully resists this dread disease is undoubted.

The fact, as the Germans put it, that "everyone is a little bit tuberculous," goes to show how easily the disease is preventable. Says Ritchie,* pertinently: "If the American people would use one-half the sum that tuberculosis is costing them each year in fighting the disease, it is probable that tuberculosis would soon cease to exist."

There are certain well-recognized local predisposing conditions, such as the paralytic thorax, anæmia of the lung-texture, collapsed air cells, all of which can be overcome successfully by suitable exercises, and thus lung resistance increased. Says Ingals,† in a paper read before this Association on the same subject as that of the present discussion: "One of the most common observations made by medical men is that the long, narrow, flat-chested individual is

* "Primer of Sanitation," by John W. Ritchie, p. 175.

† *Transactions of the American Climatological Association*, 1898. vol. xiv.

the one who is most liable to the development of tuberculosis; and we have all observed that in patients presenting this form of chest the chances for recovery are reduced to a minimum. This single observation is sufficient to suggest that systematic physical training, which would develop the respiratory muscles, expand the thoracic walls, and correspondingly increase the pulmonary capacity would be of great service, not only in preventing tuberculosis, but in curing its early stages."

The true significance of systematic physical exercises and deep breathing for their effects in increasing the vital power and resistance of the lung-texture has not been given due prominence by the profession. From the personal results obtained, I feel strongly that a growing tendency on the part of the profession to make trial of this suggestion would soon lead to its more general adoption.

The rationale of the physiologic effects of muscular exercise and deep breathing is easily comprehensible, for thus are obtained exhibitions of greater distension of the air-cells, improved circulation of the blood through the lungs whereby the localized anæmia is removed; in other words, there is brought about an improved metabolism and a greater resistance of the lung-texture. To show the beneficial effects of appropriate breathing, I quote again from Ingals' excellent article: "One of the first injunctions we should place upon the patient who has any reason to anticipate the development of pulmonary tuberculosis is that he should expand the lungs thoroughly several times every day. We must also personally inspect his mode of breathing to see that he knows how to carry out our instructions. In order to expand the lungs, the patient should be directed to draw in the abdominal walls and take a long, deep breath, while the shoulders are carried gradually backward, and the ribs and sternum elevated as far as possible; he should hold his

breath for a few seconds, and then blow it out slowly and forcibly through a small opening between the lips. In this way not only the air-cells, which can be reached by direct inspiration, are inflated, but also those at the apices and along the borders of the lungs, which otherwise might not be distended."*

Equally important for its effects upon the respiratory system is general muscular exercise. It must be taken systematically, and for the most part out in the open, as well as wisely prescribed and regulated in order to be rendered effective. At the outset of this aspect of my subject, I wish to disclaim all responsibility for the encouragement which has been given to professional athletics in recent times. On the other hand, it seems to me that professional interest should, without difficulty, be aroused to a vigorous discussion of the possibilities of muscular development with a view to securing a high standard of physical efficiency. To this end the proper use of the muscles easily shares first honours with diet and fresh air, but the former factor has received less professional attention than the two latter.

It is probable that in the presence of the local conditions mentioned above, *e.g.*, paralytic thorax, pulmonary anæmia, an implantation of tubercle bacilli may have already taken place, so that an accurate diagnosis should be attempted in all suspects by modern methods; but even though incipient tuberculosis is recognized, carefully supervised physical exercise is to be advised and encouraged whenever the maximum daily temperature is not above 99·3° F. and the effects noted.

In explanation of the beneficial effects of muscular exercise a few familiar principles may be enumerated: (*a*) The process of disintegration of muscular tissue is hastened by

* *Loc. cit.*

physical activity, and hence renewal or repair is to a corresponding degree stimulated, provided that sufficient rest be allowed during the intervals. (*b*) It is a physiological law that the working power and resistance of the individual are within certain limits, at all events, in direct relation to the activity of the metabolic processes; and muscular activity, particularly active outdoor exercise, is the principal factor in promoting these wholesome tissue changes. (*c*) It is to be recollected that not only the muscles themselves are benefited in carrying on an "internal respiratory" function, in which the ultimate exchange of oxygen and carbon dioxide takes place in the muscles, but by their action they also stimulate the circulation, both general and pulmonary, thus aiding in the elimination of the products of combustion, superfluous heat, &c. (*d*) Muscular activity brings into more active play all of the viscera; the appetite and digestion are improved, while the circulation of the blood, the brain, and nervous system are invigorated. (*e*) Of special interest as bearing upon the subject under discussion is the fact that "increased muscular activity means increased respiratory efforts, since there is greater oxidation and, therefore, a greater loss of carbon dioxide and a consequently greater demand for oxygen. It is this increased respiratory exchange in muscular activity which increases the nutrition and general health of the body."*

If it be true that the human body can resist an implantation of the tubercle bacillus when in a state of good physical development or healthy nutrition, then obviously muscular exercise in the open air holds forth promise of great usefulness, since favouring conditions and predisposing causes of this disease may thus be overcome. I need not revert to the muscular exercises most suitable to bring about a healthy

* *Loc. cit.*

condition of the system. It should ever be recollected, however, that these will depend upon the occupation, period of life, and strength of the individual at the start. For example, the work in which individuals are engaged being greatly varied, calling into action now one set of muscles and nerves and now another, it follows that recreation exercises must be similarly varied in order to bring into play those portions of the body that remain inactive during the ordinary avocations of life.

The amount of general muscular exercise necessary to the accomplishment and maintenance of sound health, required by a man of average height and weight in the prime of life, is equivalent to a daily walk of six miles on a level path; while growing lads and women, who are rated as equal to lads of sixteen, require somewhat less. Obviously, this daily quantum of exercise is needed by those whose employment is principally sedentary. On the other hand, an active outdoor life may diminish the six miles' walk quite considerably. It follows from the foregoing facts that the details connected with the regulation of the muscular exercise will vary with the individual.

In conclusion, I believe that well-regulated physical exercise is one of the strongest safeguards that we have for the maintenance of a national physique that is vital to the successful prevention of tuberculosis. Lastly, I am ready to join with those who try to limit the abuses incident to competitive sports and training.

DISCUSSION.

Dr. Otis: Some years ago I read a paper before this Association. It was here in this city I believe, somewhat similar to that of Dr. Anders's. It was, however, upon gymnastic exercise as a prophylaxis of tuberculosis, and not exercise in the open, which, of course, is much to be preferred. In the stress and strain of modern life, however, it is often difficult to persuade men to take the time for

physical exercise out of doors, particularly with that class of young men who are just beginning their life-work and are devoting all their time and energies to it; or later on, when they are in the midst of exacting duties. Time and again I have tried to persuade such persons to take some kind of daily physical exercise out of doors, but so often the answer is that they do not have the time during the day. Students in school or college, or men who are gradually retiring from more active duties, are in a more favourable position for taking the time for outdoor exercise, and they perhaps do not need it so much as the former class. There are two points with regard to physical exercise that one must bear in mind, and those are that physical exercise should be followed by rest, and that in order to obtain the most benefit from the exercise one must have a certain amount of remaining vigour—not take exercise when one is mentally or physically exhausted. When such is the case *rest* is needed, not exercise. I recall a case of a business man who came into the gymnasium exhausted from a day of hard mental work, who thought he could refresh himself by physical exercise. At the close of his class work he was more exhausted than before, and his blood-pressure was lower than at the beginning of his exercise. If one cannot take the time for outdoor exercise, then I think that carefully supervised gymnastic exercise which is made attractive, like class work to music, and which can be taken in the evening, is an excellent substitute, and will accomplish much. Whether out of doors or indoors, if one is to maintain his physical and mental integrity and prolong his life, sure it is that he must take systematic physical exercise as long as he lives.

Dr. C. A. ALTON (Hartford): I want to make one brief comment on the matter of pulmonary exercise or pulmonary athletics. It seems to me that lung expansion is an exceedingly important thing to consider in those cases that we call pre-tubercular, with no evidence but the diminished vitality of the patient to lead us to fear something to come. There is a large class of men, usually from 20 to 35 years of age, in stores as clerks. These men have a low resistance power. We take their chest measurement and find a difference of perhaps only an inch and a half between expiration and inspiration. They do not know how to expand their lungs. There is one simple method that I have not infrequently advised in cases where I feared that the slight pulmonary expansion might lead to trouble later on, and that is pulmonary gymnastics as an evening exercise. The patient starts out before going to bed, walks a block, then stands and fills the lungs as full as possible, walks a little further, and then the air is expired. This is repeated several times. These are the cases we cannot send into the mountains to walk up hill to get the same result, but they can get it at home, late in the evening after they have rested from the fatigue of the day and just before going to bed. I think if one would follow this method in a given number of cases he would be surprised at the beneficial result.

Dr. W. H. SWAN: I think this is a very important subject, and a very valuable paper, and I was very glad to see the distinction drawn between the value of physical exercise and the overtrained college athlete. I have seen a considerable number of men who in college had been in football teams and boat crews, who had afterwards broken down with tuberculosis; and, although I have no statistics at all, I have the distinct impression that they do badly. I am sure I have not seen one man who was in the college team, and was thoroughly trained, as I believe they are trained, who has afterwards done well when he had tuberculosis. In line with Dr. Otis's statement regarding rest after exercise, I think that is extremely important; and also the question of not exercising when the person is mentally fatigued. I believe that our tank of energy is all one, and if we drain it by mental fatigue and nervous fatigue we have not any left for physical exercise. I think that if the person is very tired mentally he will get no good, but harm out of it.

Dr. PERKINS: I appreciate what Dr. Minor and Dr. Swan have said in regard to the rest of children from tuberculous parents, and those who are fatigued with mental work, and I stand agreed fully with them. I think, however, that in our fresh-air schools one of the advantages we have is the exercise that is given these children, and I believe in all of our schools there should be a period of intermission and exercise. Part of the benefit is because the children are mentally fatigued. It gives their minds a rest, they can work better afterwards, they can do far better mental work after exercise, as I myself have experienced many times, and I think the most of us have felt better when we have taken a fifteen or twenty minutes' brisk walk after a hard afternoon in the office.

Dr. STONE (Boston): It seems to me that at least a start in the right direction has been made. The progress may indeed be slow, but among the moderately well-to-do people the children have made a marked advance in the past few years, with the result that the boys and girls are well developed and strong in spite of the urban conditions under which they grow up. One frequently hears mothers complaining that their young daughters are "so big"; that they wear their mother's gloves and shoes, &c. The children are well cared for and develop well. Only a little later, however, one finds these fine girls doing all sorts of foolish things to conform to fool fashions. They have become young women, and the shoes, corsets, gowns, excitement, and late hours of the present-day life too often undo the good work of the preceding years. A good start has been made for many in school life; it must be continued into young adult life. In the schools there has been a marked change for the better in the matter of fresh air ventilation. In Boston there is a general rule that between each lesson period the windows shall be opened while the class does gymnastic and breathing exercises; and also again opened at recess and before the sessions. This rule is at present

much modified by many ancient and thin-blooded teachers. Open-air schoolrooms are increasing in numbers, and are receiving more and more support from the community. At present in Boston we are preaching the open-air schoolroom gospel by an illustrated lecture at the moving picture houses, thus reaching many thousands of mothers and children. Still another advance is being made by the welfare work of some of our large stores. Recently I visited one of the new department stores, and saw the provision that was made for the rest and exercise as well as the proper feeding of the employees; all under the most favourable conditions of fresh air. These and many other straws show that the fresh-air wind is blowing favourably. A start, at least, has been made, and we must all do what we can to encourage the progress of the needs set forth in Dr. Anders's paper.

Dr. ANDERS: I would like to refer very briefly to what Dr. Otis has said with reference to the difficulties in carrying out physical exercise among clerks and business men as a class. On the other hand, it is possible to give them such directions as they can and will follow, and secure a fair amount of muscular exercise. My own method is to request such patients to walk to their work or places of business; and if it be too far, then a portion of the distance only. I often tell them to leave the trolley car or other conveyance in which they have been riding when within ten blocks or a mile of their offices, and to walk to their destination very briskly; then again at the noon hour a similar distance is to be taken. Mornings and nights they must be taught to practise calisthenics for five minutes, and if carried on systematically this proves most useful. If we can impress upon the minds of our patients sufficiently the importance of this measure, by and by they may develop a fondness for it, and would not on any account neglect it. Of course, exercise must be alternated with rest, but I thought I had placed sufficient stress upon that point in the paper. That this work should, and eventually must, begin among the children in our public schools is undoubted. As compared with the amount of attention given to mental training, surely that bestowed upon physical training, even in this enlightened age, is far too little. We want, if possible, to create public sentiment in favour of more and better-graded physical education; or, in other words, less pushing of the mind at the expense of the sinews and muscles.

THE TWO MOST IMPORTANT FACTORS IN THE PREVENTION OF TUBERCULOSIS.

By DE LANCEY ROCHESTER, M.D.
BUFFALO, NEW YORK.

The fact that the mortality from tuberculosis has diminished to a considerable degree in those localities where a vigorous anti-tuberculosis campaign has been waged shows that more cases have been recognized in a curable state and by proper methods of treatment have been cured. So far as I know there have been in the past, and there are now no reliable statistics showing the incidence of the disease, so we do not know whether there has been lessening in the morbidity from the disease or not. Personally, I believe there has not been such a lessening. For the prevention of any infectious disease it is necessary to so act upon the tissues of the body as to render them an unsuitable medium for the growth of the particular micro-organism which causes the disease or to prevent the dissemination of this pathogenic micro-organism. Examples of what can be accomplished in the former way are the vaccination to prevent small-pox and the anti-typhoid inoculation to prevent typhoid fever. Examples of the latter—the prevention of the spread of malarial and yellow fevers by the destruction of the particular species of mosquitoes which carry the germs of those diseases.

The preparation of the tissues of the body, so that it will not be a good medium for the growth of the tubercle bacillus, has been the aim of the profession for many generations. Merely to mention this fact is sufficient to show what an utter failure has been made. In the way of general building up of the tissues all the different kinds of foods, from the mineral salts to the most complex organic preparation, including fats, phosphorus, carbohydrate and protein, have been used. Improvement in environment and in habits of life has been brought about. In spite of all these measures, the incidence of the disease has not materially decreased.

Ever since it was recognized how immunity was gained in infectious diseases great interest has been manifested in, and many studies have been made of all the acid-fast bacilli in any way related to the tubercle bacillus. The only forms thus far proven pathogenic to man are the human form and the bovine form of the tubercle bacillus. Attempts have been made to find some form of the bacillus which would be used to produce immunity without producing disease.

Some of these attempts have been partly successful, in that guinea-pigs thus inoculated have lived longer than the controls, though they eventually died of tuberculosis; nevertheless, it gives ground for encouragement for further studies along this line. So far, however, all attempts to find any agent which will act as a specific preventive producing an active immunity like the anti-typhoid inoculation and the vaccination against small-pox have failed.

Thus we see that the attempt to put the tissues in a state in which they will not be a suitable medium for the growth of the tubercle bacillus, either by building up the general resistance or by stimulating a specific resistance has failed.

Thus we are brought to the conclusion that we must in some way control the dissemination of the tubercle bacillus. As said earlier, the only two forms of the tubercle bacillus

which are pathogenic for man are the bovine and the human forms of the tubercle bacillus. This being so, we must recognize that the two sources of these organisms are tuberculous cattle and the discharges from tuberculous lesions caused by the tubercle bacillus, of which, of course, pulmonary tuberculosis is the chief.

The detection and destruction of tuberculous cattle will, of course, eliminate the extension of the bovine form of tuberculosis to the human race, and the passage and enforcement of laws to this effect should be persistently urged. In this way one of the two great factors in prevention will be made effective.

The other factor, which I think is much more important, I called to the attention of this Society in October, 1903. It is the compulsory segregation of the advanced cases of pulmonary tuberculosis.

Those of us who have charge of hospitals for advanced cases know with what difficulty they are controlled even in the sanatorium, that they are careless about their sputum and about their general hygiene, that it is almost impossible to make them cover their mouths with gauze during the act of coughing, and very difficult to make them understand that the piece of gauze should be used only once, and should never be put into the pocket or under the pillow.

Even if they do follow out directions pretty well while in the hospital, when they get to feeling a little better they leave the institution and go outside to work, usually living in cheap lodging-houses, often a different one each night. They cough and spit and take no precaution, thus infecting their surroundings and their fellow-lodgers. At present I think that most cases of pulmonary tuberculosis are reported to the health department. That department should by law be given more power in regard to these cases. Every case should be investigated, and if unable or unwilling to properly care for

himself at home, should be committed to one or other sanatorium for incipient or advanced cases, to stay there until cured or dead.

In our county tuberculosis hospital we have had great difficulty in maintaining proper discipline. It is impossible to keep those patients, who are up and able to be out of doors, on the hospital grounds; as a result they not infrequently go down town, visit saloons, undoubtedly infect them, and return to the hospital in a day or two in an intoxicated condition. It is wrong to refuse to take them in, as outside they are a danger to the health of the community. To take them back to the hospital prevents any true discipline in the hospital, and the offence is committed over and over again.

If they were committed to the hospital as, for example, the insane are committed to the State Hospital, they could be compelled to remain on the hospital grounds, and would be properly punished for infraction of rules.

It may be urged that it is an unnecessary hardship to compel a patient to die in an institution, excluding him from the privilege of passing his last days in the bosom of his family. To a very limited degree this is true, but in tuberculosis the end is seldom sudden, and the members of the family can be notified and allowed to be present at such a time.

In the case of other great plagues the isolation is more complete, and the friends are not allowed to be present even at the last. So the procedure is not unprecedented, and the good to come to the entire race from such sequestration kept up persistently for ten or fifteen years would far outweigh the slight psychic disturbance of a few. The bodies of all dead from tuberculosis should, of course, be cremated.

Instruction in regard to destruction of sputum and fæces, in regard to precaution as to personal contact with others, and in regard to disinfection of apartments lately

occupied by the tuberculous, are all of value in checking the spread of the disease, but it is my opinion that some such plan as has been outlined, of compulsory reporting of cases and compulsory removal of suitable patients to their proper sanatoria, will be much more efficacious than all the other procedures in ridding us of the great white plague.

DISCUSSION.

Dr. OTIS: I would like to ask one question of Dr. Rochester, as to why all bodies of tuberculous subjects should be cremated.

Dr. ROCKHILL (by invitation): We have quite a large sanatorium of 330 beds in the City of Cincinnati, and we are spending $360,000 for improvements, and among other buildings we are putting up a detention ward for thirty males and five females. The health officer has the opinion of the city solicitor that he can detain and have confined vicious tuberculous patients, so we have already two rooms that we use for that purpose. We confine three or four cases there, although we are not able to keep them there for an indefinite period; but even with the three or four beds that we have, and the confinement of the patients in those rooms, the moral effect is tremendous. I think that with a ward where we can put away thirty such cases the moral effect will be very beneficial.

Dr. C. L. MINOR (Asheville, N.C.): The subject of this paper is one that has interested me for a long time. Whatever we may have accomplished in the crusade against tuberculosis, the question of the handling of the criminal and ignorant poor is an almost insoluble problem. The intelligent and manageable poor can be taught in proper institutions, but the criminal and ignorant will not, or cannot, learn and are the great source of the spread of the disease. The effect of segregating such advanced cases has been shown very clearly by Dr. Newsholm, of England, and need scarcely be referred to here. Ordinary treatment in ordinary sanatoria cannot be expected to do these people any good, nor to render them harmless to those among whom they live. Since then they are an unavoidable danger to the community, I believe the community has a right to segregate them under police control, just as it has the right to segregate small-pox and other contagious disease cases, and until this is done a large part of our efforts towards eradication of tuberculosis are wasted.

Dr. OTIS: I want to ask if I am not correct that the human tubercle bacillus will live only at a temperature of 37° C.

Dr. DUNN: I had an interesting experience recently that I think

will, perhaps, be worth while relating, even if it is out of order. Some years ago I had occasion to set aside some cultures. The culture was implanted in March, 1907. Four weeks ago I found that culture, and I thought I would simply see whether it was still alive or not, and that culture grows with facility; it is one of the most rapidly growing cultures.

Dr. ROCHESTER: Dr. Otis asked why I said that the bodies of people who died with tuberculosis should be cremated. I feel that all bodies should be cremated any way, but that is another question. The reason I said this in regard to tuberculosis is that the tubercle bacilli have been demonstrated in the bodies of earth-worms, and it is possible that we may have a spreading of tuberculosis through these worms getting upon plants and spreading through lettuce or some other food which may be eaten. It is a mere possibility, but it is a possibility to consider. I thoroughly believe in cremation. There is one other view on which I wish to very decidedly express myself in regard to the detention ward; I am very glad to hear Dr. Rockhill state that they have already established that detention ward in Cincinnati. We are about to try it in Buffalo. I have in mind just one case which illustrates the seriousness of letting patients out to go home to die. I had a man of advanced tuberculosis some years ago who finally went home to die. He had a wife and five children. Four of these children and the wife have since died of tuberculosis. He went home to die and they died too.

Dr. BALDWIN: I think these statements will only illustrate what was said here, that the laboratory results are sometimes fallacious. It has been proved that these earth-worm bacilli are not true tubercle bacilli. It is now known that they are saprophytic bacilli, that grow at cold-blooded temperature, and the earth-worms are not of any danger to the human being. The turtle bacillus is exactly the same kind.

"AURICULAR FLUTTER," WITH A REPORT OF THREE CASES.

By DR. FRANK TAYLOR FULTON.

PROVIDENCE, R.I.

In 1911 Jolly and Richie [1] reported a case of Adams-Stokes disease which had been under observation for a period of six years and which showed, in association with the complete heart-block, a very rapidly-beating auricle, the rate of the latter varying from time to time, but usually from 270 to 300 per minute. This condition they called auricular flutter. In adopting the use of the term they refer to the work of McWilliams [2], who, years ago, observed that the application of a faradic current started the auricle into a rapid flutter. In his account he stated that the contractions originated in the stimulated area and extended rhythmically and co-ordinately throughout the tissue. In 1909 Hertz and Goodhart [3] reported a case with an auricular rate of 234 and a ventricular rate varying from 72 to 120. In this case the irregular and varying ventricular rate depended upon a partial heart-block, although this was not recognized at the time. Lewis [4] has since written a very comprehensive article on the subject, in which he reports eight cases which have come under his own observation and eight other cases which have appeared in the literature from time to time, and which, apparently, are all types of this disorder. Hume's [5] case, no doubt, is the same mechanism.

Auricular flutter when first described was considered a very rare condition. As a matter of fact it is probably comparatively common. The reason for its apparent rarity is that it is impossible to detect these extreme accelerations except with the use of graphic methods, and unless a routine examination by these methods is carried out many of the cases will escape recognition. The condition is an abnormal cardiac mechanism characterized by a rapid, rhythmic, co-ordinate contraction of the auricle, the rate usually being somewhere between 200 and 300 per minute. A notable characteristic is the constancy in the auricular rate, any change due to posture or exercise being almost entirely negligible. Some degree of heart-block is usually present; consequently the ventricular rate is slower and may be regular or irregular.

It is a widely-accepted belief that the normal cardiac impulse is brought about by a stimulus which arises in the sino-auricular node, sometimes called the pacemaker, situated near the mouth of the superior vena cava. This node gives rise to impulses in a healthy adult at the rate of about 72 to the minute, the rate being subject to wide variation as the result of exercise, emotion, fever, and various other causes. The details of the mechanism which controls the rate are by no means clearly understood—whether the ordinary accelerations are due to diminished vagus tone or to direct stimulation through the sympathetic or to some other cause, it is not easy to prove; but, at any rate, it seems clear that all these simple accelerations are under the control of this mechanism, whatever it is, and that all the impulses come from the normal point of origin. These have been called tachycardias of a physiological type, because they are simply the normal phenomenon exaggerated.

In auricular flutter the predominating evidence is that the origin of impulse is ectopic; that is to say, some other

point than the normal pacemaker has taken on the function of stimulus production. It is well known that the ordinary premature auricular beat is the result of some pathological, usually ectopic, impulse formation. If a series of these beats arise in quick succession the condition is recognized clinically as paroxysmal tachycardia. This is usually characterized by a rate of 110 to 200—rarely over 180. If, however, this abnormal focus in the auricle gives rise to impulses at a rate greater than 200, the condition has been called auricular flutter. This may seem an arbitrary division, for etiologically and pathologically it apparently does not differ from the simple paroxysmal type, but clinically it differs sufficiently so that it is considered advisable that it be classed in a separate category. It has been found, also, to have a close relation to auricular fibrillation, and when one considers the pathology and mechanism it is easy to see how readily a condition in which there is one abnormal point taking on the function of stimulus production, might pass into a condition in which many foci have appropriated that function.

Some of the reasons for believing that the stimulus which gives rise to this action is ectopic in nature are: First, the already mentioned close relation between this and the isolated premature auricular beat, simple paroxysmal tachycardia and auricular fibrillation. Secondly, that the auricular complex, as recorded by the electro-cardiograph, differs essentially from those which rise from the normal pacemaker. Thirdly, that the rhythm is not under nerve control, as is the normal rhythm, being practically unaffected by posture, exercise or nerve stimulation.

The rate of the auricular beat varies considerably. In the cases collected by Lewis it has varied from 200 to 330 per minute. As already mentioned, this rate is remarkably constant, although observations made at long intervals of

time show some variation in the rate. Observations made on the same day or on succeeding days usually show but little, if any, change.

The ventricular rate is usually slower, due to some degree of heart-block. The most frequent condition is a 2 : 1 ratio, in which the ventricle responds to each alternate auricular contraction. However, there may be any variation, and instead of 2 : 1, the response may be at the ratio of 4 : 1. In either instance the ventricular rate is regular. If, however, as it is now known frequently occurs, the responses are mixed so that a 2 : 1, 3 : 1, 4 : 1, 5 : 1, &c., may be present, the pulse becomes entirely irregular and may be with difficulty distinguished from auricular fibrillation. The ratio of response may vary with exercise, so that a patient who lying down would have a regular pulse at the ratio of 4 : 1 might when standing have a regular pulse with a 2 : 1 response, or a patient with a regular pulse with a ratio of 2 : 1 while active might have an irregular pulse because of mixed responses when at rest. In most of these cases the original grade of heart-block is increased by digitalis or strophanthus, which suggests that there is probably some impairment of muscle bundle conduction, for it is believed that these drugs have little, if any, influence on the conduction in the normal heart. Following the use of these drugs the regular 2 : 1 response may be converted into a condition of mixed responses of 2 : 1, 3 : 1, 4 : 1, 5 : 1, &c. An interesting fact pointed out by Lewis is that in the majority of instances these cases will pass from a 2 : 1 to a 4 : 1 ratio rather than from a 2 : 1 to a 3 : 1, and if the ventricle is irregular it is likely to consist of mixed 2 : 1 and 4 : 1 periods, though, as Case 2 of those I am reporting shows, this is not always true. Isolated 3 : 1 or 5 : 1 periods may occur, but they are comparatively rare, and when a 3 : 1 ratio occurs successively the periods are usually short.

The Polygraphic Tracings.—The first case of Jolly and Richie in which there was complete heart-block and in which the jugular tracings were taken by a Knoll-Hering polygraph showed distinctly the auricular waves. On the other hand, in many cases the jugular tracing is of no value in the diagnosis and may even be of such form as to be entirely misleading. In fact the venous pulse in a 2 : 1 ratio is likely to be of the ventricular type. Occasionally, when the ratio is 4 : 1, one may get an auricular wave toward the end of the ventricular pause, and if, as sometimes happens, there is an unusually prolonged pause there may be a series of auricular waves toward the end of it. Sometimes, as in two of my cases, the auricular waves are so prominent as to seriously complicate the c waves, and in the attempt to identify the latter one may get a clue to the condition. The radial tracings are often of much more value in the analysis than the jugular, and a close examination of the arterial pulse curve alone may enable a positive diagnosis to be made. The points to be borne in mind as given in detail by Lewis are: First, that alternation is commonly present; second, that the strength of the beats is substantially influenced by the preceding pauses. This is true, however, only when the pauses are too short for the ventricular contraction to be of maximal efficiency; third, that in the heart-block of flutter there is considerable variation in the As-Vs interval which modifies the expected pauses—a long pause is followed by a shortened conduction interval, and a short pause by a lengthened conduction interval; fourth, that the weak beats are likely to be preceded by a relatively long presphygmic interval. All these factors must be considered and contribute to the difficulties of the analysis. Usually when the ratio of ventricular response is irregular there is a definite grouping of beats, so that groups of three or four beats of irregular lengths may be repeated over and over

again; but in some instances the ratio of response is so mixed that the condition is distinguished from fibrillation with difficulty.

Treatment.—The general management of the case depends upon the symptoms. If there is evidence of cardiac failure, of course rest in bed is of paramount importance. The latter is just as beneficial in this condition as in any other in which the heart muscle is overtaxed. This was shown in Case II, who had no medication for four days after admission to the hospital, but whose symptoms improved progressively from the time of admission though the pulse remained unchanged. Ordinary sedatives should be used, as indicated in the individual case.

The particularly beneficial drugs, however, are digitalis and strophanthus. In several of the reported cases, during the administration of digitalis the flutter has passed into fibrillation, and when the fibrillation has disappeared the normal rhythm has become re-established. Lewis explains this by saying that fibrillation seems to submerge the abnormal fast rhythm which may not recur when the fibrillation passes off. As has already been stated, digitalis and strophanthus usually slow the heart by increasing the grade of heart-block, so that these drugs may act beneficially in either of these two ways, namely, by causing fibrillation, which may subside leaving the rhythm normal, or by slowing the pulse through increased heart-block.

In Case II a grain and a half of digipuratum was given four times a day for eight days. At the end of that time, as indicated by the tracing taken on March 31, there was some increase in the grade of heart-block causing a slowing of the pulse with some characteristic irregularity. What the dominant mechanism was during the next month is a matter of conjecture, but presumably flutter was persisting all this time, and on April 29 a tracing showed what appeared to

be a 4:1 block. The amount of digitalis the patient had been having at that time is not definite. The patient was put on digitalis subsequent to this, and a tracing taken on May 15 showed that the auricle had gone into fibrillation. The date on which this occurred is not known. Whether the fibrillation will be permanent or whether the normal rhythm will become re-established, as frequently happens, cannot at present be stated.*

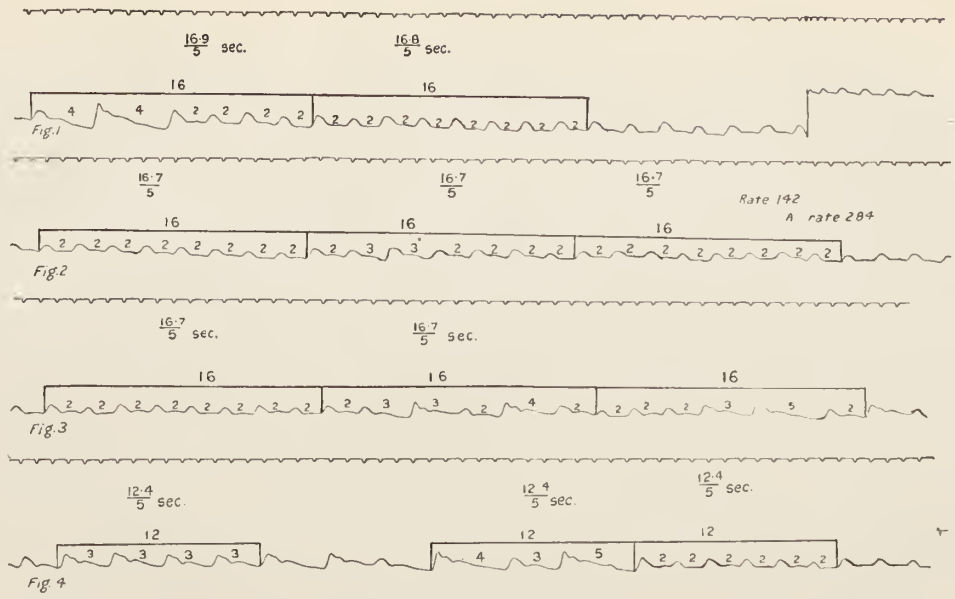
I shall report three cases, two of them showing typical polygraphic tracings which, when analysed, leave no doubt that the mechanism present is that of auricular flutter. The third is not so typical, but the evidence that it is a case of flutter is quite convincing. I have not been able to get electrocardiograms, but have had to depend entirely upon the polygraph.

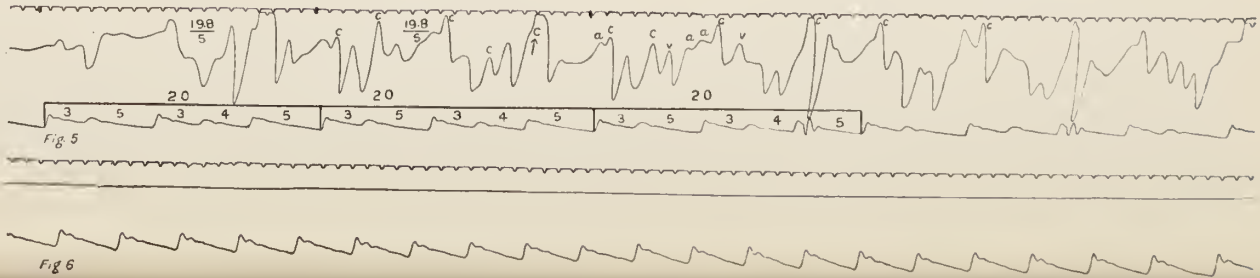
Case 1.—E. C., aged 48. Was admitted to the Rhode Island Hospital, November 20, 1912, and came under my care January 1, 1913. Patient is a native of Rhode Island, and a jeweller by occupation. His family history is unimportant. He gives no history of any previous illness which might have any bearing on his present condition. He drinks considerable alcohol habitually and occasionally to excess. A year before admission he began to get short of breath on exertion. About five weeks ago his legs began to swell, and shortly before admission the swelling had extended to his hands, face and genitals. He had to get up at night to void urine occasionally. He was at work until within a week of his admission to the hospital. On admission there was slight swelling of all of the extremities and of the genitals, with some œdema of the abdominal wall, but no definite signs of fluid in the abdomen. There were moist râles in the bases of both lungs behind. Heart apex was in the fifth space, 8 cm. from the mid-line, and the organ was not definitely enlarged. Sounds were regular and clear,

* The auricle was still in fibrillation, August 9, 1913.

the aortic second slightly accentuated. The pulse was regular, of good volume and tension. Examination of the urine showed a trace of albumen with a moderate number of hyaline and granular casts. The quantity was rather scanty. The functional test with phthalein showed 20 per cent. efficiency. Patient had a good deal of dyspnœa, which came on periodically, some days being much less marked than others. It was relieved with morphine. His condition remained the same for a number of weeks, except that the œdema of the extremities was increasing and the dyspnœa was perhaps more marked. His pulse was noted frequently and was always regular. At the time of the regular visit, on the morning of February 10, it was noted to be rapid and irregular. A tracing of the arterial pulse was taken at that time, portions of which are shown in figs. 1 to 4 inclusive. These tracings show considerable stretches of an absolutely regular pulse at the rate of 143 per minute. Interspersed here and there in the tracing are irregularities of greater or less length. The evidence afforded by analysis is that the auricle is beating at the rate of 286 per minute and that the regular strips are a 2 : 1 response of the ventricle, while the irregularities are markedly irregular, the ratio of 2 : 1, 3 : 1, 4 : 1, 5 : 1 all being present.

While the dyspnœa did not seem any more distressing with the onset of this irregularity, the œdema did increase more rapidly after this rhythm was established and was relieved only by the use of Southey's tubes. In a tracing taken February 20 (see fig. 5) the irregularity was quite different—at this time a very definite grouping being quite persistent. For example, a group of five beats of varying lengths is repeated over and over again, the entire length of the group being $1^{\circ}\frac{8}{5}$ seconds. The estimated ratio in these groups is 3 : 1, 5 : 1, 3 : 1, 4 : 1, 5 : 1, which would give an auricular rate on this day of about 308. Occasionally





(Case I.)

Figs. 1, 2, 3 and 4 are all a part of the same arterial tracing taken on the day the irregularity was first noticed. The short runs where the pulse is regular is a 2 : 1 block. The irregularities show ratios of 2 : 1, 3 : 1, 4 : 1, and 5 : 1. At one place in fig. 4 are four consecutive beats at the ratio of 3 : 1. The calculated auricular rate is 284.

Fig. 5 was taken ten days after the onset. A group of five arterial beats of varying lengths recurs with great regularity. The estimated ratios here are 3 : 1, 5 : 1, 3 : 1, 4 : 1, and 5 : 1, giving an auricular rate of about 308. This, however, cannot be absolutely proven from the tracing. The jugular pulse shows chiefly ventricular waves.

Fig. 6 was taken just after the normal rhythm was re-established. It shows some sinus arrythmia due to Cheyne-Stokes respiration.

there is a break in this group. This analysis of fig. 5 would seem most probably correct, though the evidence is not absolute.

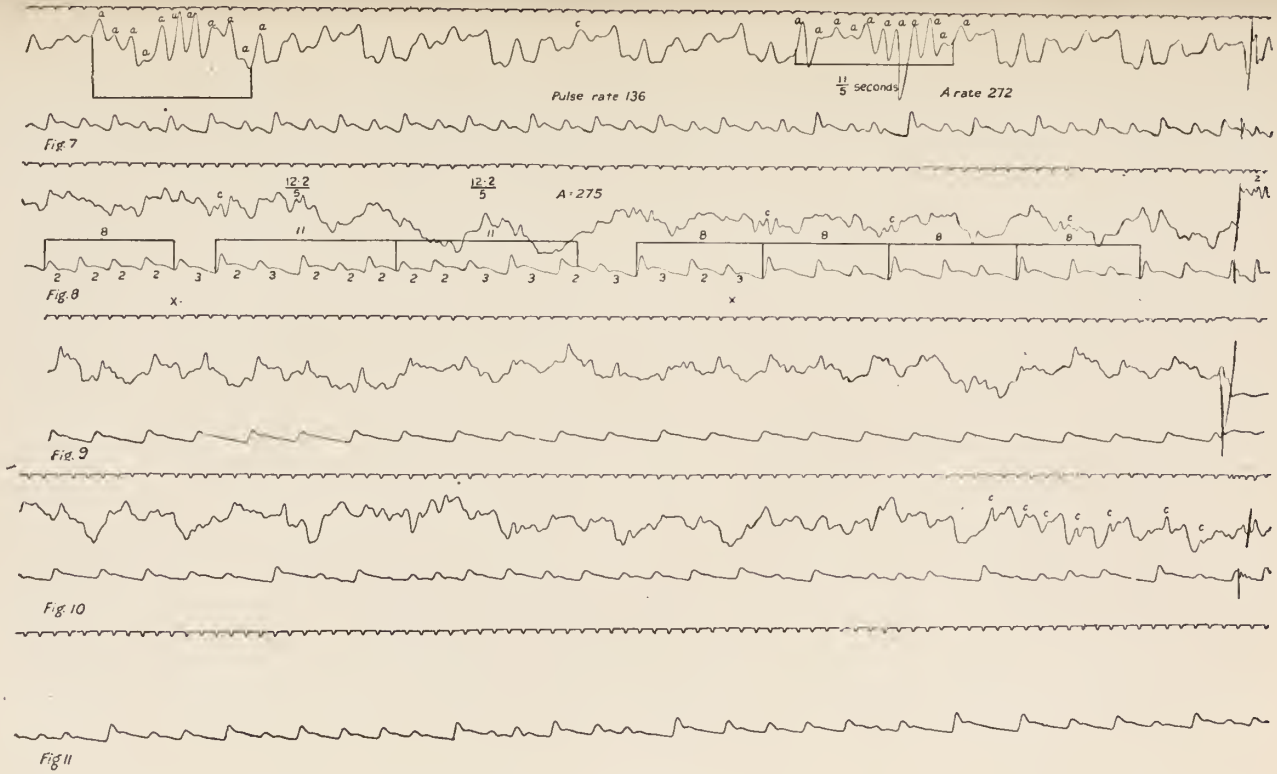
Patient was given tincture of digitalis mxv t.i.d. from February 20 to March 15. The irregularity continued until March 10, when it was noted at the morning visit that the pulse was regular. Tracing taken at that time showed the rhythm to be normal except for its modification from the Cheyne-Stokes breathing (see fig. 6). The patient is still in the hospital without any very material change in his condition. At times his dyspnœa is marked and at times the Cheyne-Stokes breathing is marked, but there are other times when both of these are entirely absent. The œdema is still quite considerable, and although it cleared up materially with the return to the normal pulse rhythm, it is still necessary to remove some of it by Southey tube drainage.

Summary.—A man, aged 48, suffering from progressive chronic nephritis with paroxysmal dyspnœa and considerable œdema, suddenly developed an irregular heart action after nearly three months in the hospital. This lasted for a month, when the rhythm suddenly became normal. There were no noticeable symptoms with the onset or end of the attack, but the œdema was considerably more marked while the condition persisted. Cheyne-Stokes breathing was present at the time of the return to the normal rhythm. Digitalis in moderate doses of the tincture was being administered during most of the time of the irregularity. The tracings indicate that the irregularity was due to a very rapidly-beating auricle associated with irregular ventricular responses.

Case 2.—W. K., aged 54. Was admitted to my service at the Rhode Island Hospital, March 17, 1913. Patient was a native of Rhode Island and a jeweller by occupation, and came in complaining of shortness of breath and swelling

of the legs. The physician who attended him before admission said that for a month his dyspnœa had been very marked, and that it was with difficulty that he could go up even a slight incline. The trouble had been coming on gradually for a year, but in November, 1912, he had an attack of bronchitis with a persistent and troublesome cough which aggravated the condition so that there was increased shortness of breath, some swelling of the legs, and considerable dizziness upon exertion. He had to get up at night five or six times to void urine and was troubled a good deal with constipation. His general health, he stated, had always been good, although he had had hay fever in the summer for a number of years. On admission there was a good deal of dyspnœa with orthopnœa, some signs of a slight amount of fluid in the right chest and râles with a distinct pleural friction. The heart was slightly enlarged and action very rapid but regular. There were no murmurs. His blood-pressure while in the hospital varied from 160 to 190. There was marked œdema of both legs with some dermatitis. The amount of urine averaged from 900 to 1,200 c.c. with a specific gravity from 1·009 to 1·012, showing a slight trace of albumen, but no casts.

The heart action was so rapid as to attract attention at once and a polygraph tracing was made. It showed a rate of 138 per minute with marked alternation of the pulse. Patient, at first, received no medication. Two days later another tracing was taken showing a pulse-rate of 136 and still with marked alternation. The venous tracing on this day showed in places auricular waves at the rate of 272 per minute (see fig. 7). On March 22, four days after admission, a third tracing was taken showing the pulse-rate of 137. Patient had been in bed during the four days he had been in the hospital, but was at this time allowed to get up and was given some exercises at the side of the bed and a



(Case II.)

Fig. 7.—Two days after admission. The pulse is for the most part regular at a ratio of 2 : 1. At two points it suggests a 1 : 2 and 3 : 1 ratio. The auricular rate is 272. Alternation of the pulse is well marked.

Fig. 8.—After eight days of digitalis. Showing mixed responses of 2 : 1 and 3 : 1 ratio, with short runs of 2 : 1. Patient left the hospital and was not observed for a month.

Fig. 9.—Tracing made April 29, one month later. The pulse was regular, 82 to the minute, unaffected by posture and exercise.

Figs. 10 and 11.—Tracings made May 15 showing fibrillation.

tracing taken immediately afterward. The rate was absolutely unchanged. On March 23 the patient was given digipuratum (1½ gr.) four times a day. This was continued until March 31. Pulse was observed repeatedly and was always found to be regular with the rate practically unchanged. The first irregularity was noted on March 31, after eight days' treatment with digitalis. A tracing made on that day shows a very interesting condition (see fig. 8). There are short runs of beats of the same length as had been present during all the time when he was under observation, but between these runs are periods of irregularity. Considering the condition while the pulse was regular to have been a 2 : 1 heart-block, as evidenced by the venous tracing, analysis of the irregular curve shows a heart-block of a 2 : 1 ratio in the regular strips, and mixed 2 : 1 and 3 : 1 ratios in the irregular strips. The irregularity is usually made up of groups of three beats, one of which is at the ratio of 2 : 1, the following two beats at the ratio of 3 : 1. In other words, when the pulse is regular there are four ventricular beats for every eight auricular beats, while in the irregular periods there are three ventricular beats for every eight auricular beats. Patient left the hospital on March 31, the day on which this tracing was made. He was not seen again until the afternoon of April 29. He had gone to work and had been able to work for a week. His old symptoms, however, returned, so that he had to give up. The œdema on this date was again quite marked. He was sitting up in a chair and had spent several nights sitting up, not being able to sleep. He was having slight attacks of paroxysmal dyspnœa. Cheyne-Stokes breathing of moderate type was present. Patient was able to move about the house without much evidence of shortness of breath. A tracing was taken which showed a regular pulse. Rate 82 to the minute with slight alternation (see fig. 9). The

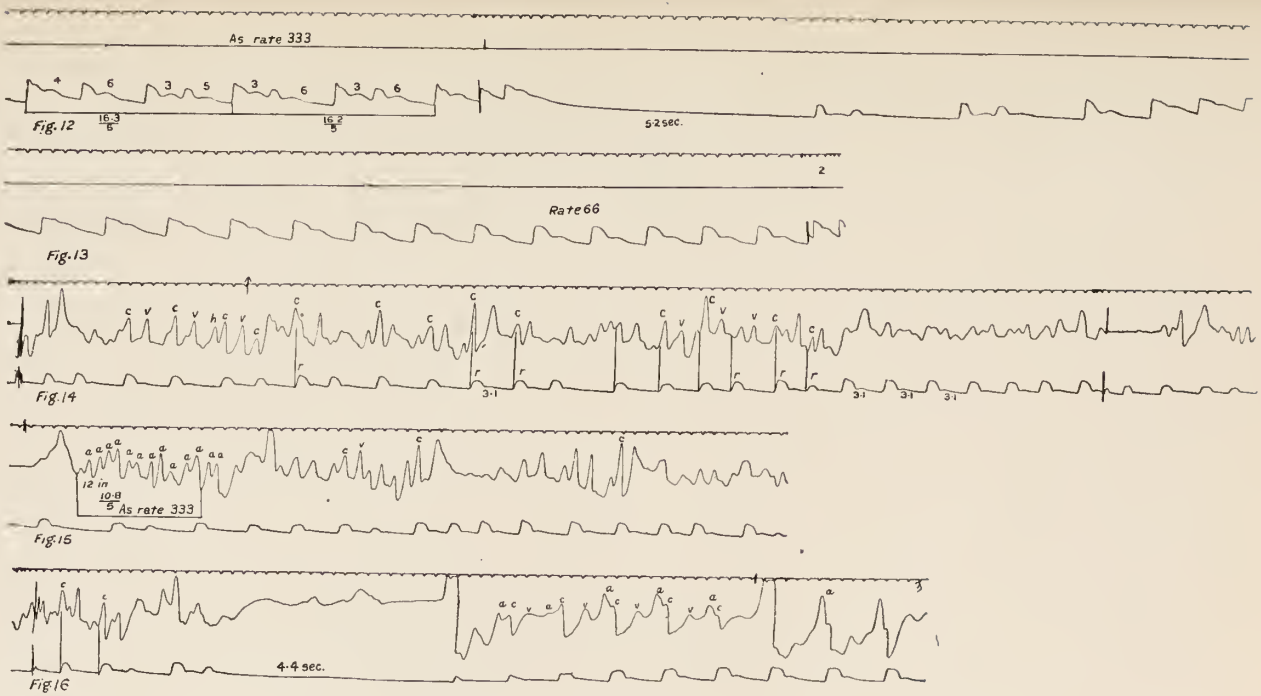
tracing was continued at intervals for about an hour, but at no time was there any irregularity whatever. The rate was absolutely constant. The patient twice got up from the chair, walked about the room, and a tracing taken immediately after showed no change in the rate. The jugular tracing was not very satisfactory, and while there were some extra small waves, there was nothing characteristic enough to make it absolutely sure whether the flutter is still persisting or not. The absolutely persistent rate unaffected by exercise suggests very strongly that the condition was still present and that there was a 4:1 block. Urinalysis; sp.gr. 1·020, trace of albumen with a number of small hyaline casts. Patient was again seen on May 15, and a tracing showed that the auricle had passed into fibrillation (see figs. 10 and 11).

Summary.—A man, aged 54, with gradually increasing dyspnœa and œdema for a year comes under observation with chronic nephritis and an enlarged heart in stage of decompensation and a pulse of 136 to 138 uninfluenced by posture or exercise. Auricular waves are present in the venous tracing at the rate of 272 per minute, indicating a 2:1 block. With rest in bed improvement was steady, but the pulse was not slowed until after eight days of digitalis, when it became irregular, the latter being due to an increased grade of heart-block. Six weeks later the auricle was found to be in fibrillation.

Case 3.—H. F. H., aged 70. Patient is a retired business man, who was first seen seven years ago. He had been a man prominent in affairs, but had a nervous breakdown at about the age of 60 and had given up business. About the same time (ten years ago) he began to have attacks of palpitation of the heart. These attacks would last from one or two hours to perhaps twelve. He was first seen in one of these June 4, 1906. The attack had come on late in the afternoon after nine holes of golf. When seen he was

somewhat apprehensive, the pulse was very irregular and the rate 100. There was no shortness of breath, but a sense of pressure in the præcordium and a slight tendency to cough. He went to bed and slept well. The following morning the pulse was 84 and regular. The physical examination showed nothing which would seem to have any definite bearing on the attacks. There was slight emphysema of the lungs. Heart was not definitely enlarged. Blood-pressure 100. Arteries hardly so thickened as usual at that age. Urine showed a few small hyaline casts, but otherwise practically normal. The second attack in which he was seen on September 1, 1906, came on apparently as the result of some disagreeable news. Heart was irregular as before, and the rate 100. His statement at that time was that these attacks were occurring every month or two, but he was seen in them only occasionally. The third attack on which notes were made was on November 30, 1908. This came on while in the bath-room in the morning. The explanation given by the patient was that he had had some indigestion the night before, with considerable gas in the stomach and intestines. The pulse was irregular as before, but in a couple of hours was regular and 70.

Patient was seen from time to time, but no note was again made until July 25, 1911, on which date he was seen just following an attack of palpitation which occurred the night before, and which disappeared before morning. He said then that he was having attacks about once a week. They would come on suddenly and leave suddenly. In the meantime, however, as far as his heart was concerned, he was not troubled, and was able to climb hills and stairs easily. Patient continued to have these attacks of palpitation at frequent intervals. Early in 1912 he was given $\frac{3}{4}$ gr. powdered digitalis three times a day for a week at a time, and would then omit it for a week. He continued that



(CASE III.)

FIG. 13 is a continuation of fig. 12. This tracing was started during an attack on October 8, 1912. A preliminary arterial record was being made before attempting the venous curve, and it was at that time that the normal rhythm became re-established. At the time of the transition from the abnormal to the normal rhythm there was a standstill of the heart for 5·2 seconds. Just before the transition is a period of definite spacing from which the auricular rate might be about 330.

FIGS. 14, 15 and 16 are all parts of a tracing during an attack on October 15. They show the transition from the abnormal to the normal in both the arterial and venous curves. They show in many places that what would seem to be the c waves do not coincide with the corresponding upstroke in the arterial curve. The jugular curve also shows many accessory waves besides the c and v waves which are at the rate of about 330 a minute.

quite faithfully throughout the year 1912. He was much better, and was quite free from attacks from February until September. Between September 20 and November 8, 1912, he had five attacks, and has had none since.* In four of these attacks I saw him, and in two of them I was able to get a polygraph tracing, in each of which the transition from the abnormal to the normal rhythm is shown. The tracings were examined only casually, and the condition considered one of paroxysmal fibrillation. Because the tracings had rather more than usual interest as showing the transition from the abnormal to the normal rhythm, they were sent to Dr. Lewis, who made a careful analysis, and ventured the statement that he believed the condition a case of flutter with extremely complex responses, giving as his very convincing reasons: First, the variable relation between the arterial upstroke and what was apparently the coresponding c upstrokes; second, the numerous waves in the jugular tracing in addition to the c and v waves; third, a tendency to spacing, which is very definite just before the long pause in one of the radial curves, and, in addition, the frequent recurrence of beats of the same length; lastly, that the rate of waves in the venous curves in certain stretches is about 330 per minute, too slow for fibrillation, and just the rate calculated for flutter.

Summary.—A retired business man, aged 70, with neurotic tendencies, under observation for seven years, has had frequent attacks of palpitation for ten years. These followed a nervous breakdown. The attacks come on at irregular intervals every few weeks, and are characterized by a slight sensation of oppression in the præcordium, apprehension, tendency to cough, and an irregular pulse of

* He had an attack lasting about five hours on August 3, 1913. None occurred in the interval between November 12 and August 3.

about 100 per minute. There is no marked disturbance of the circulation. The onset and end of paroxysms are generally definitely recognized unless the end comes during sleep. Between attacks there is no evidence of any cardiac or renal insufficiency. Patient thinks they usually come on as the result of some emotion or indigestion associated with gas. He has been on small doses of digitalis for about fourteen months. During that time he had five attacks between September 20 and November 8, 1912. He has had no others. Four of these were observed, and in two a polygraph tracing was obtained showing the transition from the abnormal to the normal rhythm. This transition is marked by a very long standstill of the heart, in one instance more than five seconds. Twice at the change patient has mentioned a feeling of faintness and a sensation of things growing black.

The pulse tracing is very irregular, but there is occasional evidence of spacing, and the c waves in the venous curve are complicated by a waves, so that often the former cannot be identified. There are occasional runs of auricular waves at the rate of about 330 per minute.

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DISCUSSION.

Dr. HIRSCHFELDER (Baltimore, Md.): I am very much interested in the cases which Dr. Fulton has reported, as I have for a number of years been interested in these conditions upon the border line between the normal rhythm and auricular flutter. In the cases which Dr. Fulton has reported he gave us some very interesting examples

of auricular fibrillation block, but we cannot always be certain in the tracings, on account of the spacing, whether we are dealing with a block or with a sudden change in the rate of the auricle itself. One sees that very well exemplified if one changes or alters the high tense of irritability of the auricle in an animal by faradizing, by stimulating it with faradic stimulant, as, for example, by applying it to the top of the auricular appendix. The state of flutter and the state of fibrillation are apparently a quantitative relation as regards irritability rather than qualitatively different. That is, if one makes use of a graded stimuli one finds that with the mildest stimulus to which one can get response one obtains occasionally extra response. If one stimulates with a still greater intensity one obtains a sudden doubling of the rate. I was particularly interested in this question when I first began to investigate the condition in 1906, because at the time the dominant theory was the theory of August Hoffman. As I say, if one stimulates the auricle with faradic stimuli which are far too frequent to give rise to response to the individual stimuli, one obtains a gradation from extra systole to a paroxysm, but not an absolute sudden doubling of the rate, and upon cessation of the stimulus one finds a sudden return of the auricle as well as of the ventricle to a normal rate. [Remarks uncorrected.—Ed.]

Dr. FULTON: I was very glad to hear Dr. Hirschfelder's remarks. There were a good many points which it was impossible for me to bring out in the time allotted. I emphasized particularly the analysis of the radial curve, because I am convinced that many cases of this condition can be diagnosticated without any mistake from the radial curves alone. I admit, as he says, that there are other conditions which might bring about a somewhat similar appearance, but with careful analysis I think there is no question but that the radial alone is of great value in making the diagnosis, and the reason that it is of value is that the cases have been studied with the electrocardiograph and the polygraph, so that the tracings which are so clear with the electrocardiograph may now be identified by the use of the other instrument. The question of extra systole is of course one to be considered. I do not believe that if Dr. Hirschfelder had an opportunity of analysing the cases I have that he would think there is any possibility of these tracings being produced by extra systoles. The condition is closely related to fibrillation, the essential difference being that in flutter the stimulus production is confined to one abnormal point, while in fibrillation it occurs in a number of points in the auricle.

ARTIFICIAL PNEUMOTHORAX IN THE TREATMENT OF CHRONIC INFECTIONS OF THE PLEURA AND LUNGS.

By CLEAVELAND FLOYD, M.D.
BOSTON, MASS.

Among the many complications associated with pulmonary tuberculosis, none are productive of greater physical changes within the chest that vitally influence the termination of the affection than spontaneous pneumothorax. The occurrence of partial or complete collapse of the ruptured lung is rapidly followed by a modification of the respiratory sounds in both sides of the chest and acute cardiac strain, with dilatation of the right ventricle. The immediate result, where the sound lung and right heart cannot care for the demands made upon them, is a fatal one. More often than otherwise, however, the phthisical lesion, through its stimulation of fibroid tissue and pleural adhesions, may effectually limit the extent of the pneumothorax or resist its rapid advance, and the circulatory changes are brought about with sufficient slowness to allow of complete compensation. A moderate body of air in the pleural cavity may bring about more or less complete collapse of one or more lobes, a marked lessening of the flow of blood and lymph and cessation of respiratory activity. This series of changes is followed in corresponding sequence by the diminution of absorption of

toxic products, as a result of rest of the diseased lung and the expedition of fibrous tissue formation. The small or transient pneumothorax could hardly be expected to bring about any decided change in the condition of the infected lung, but when its force is exerted over a considerable area for some time the result is beneficial. Carlson, Adams, Sale and other observers have noted the good results which not infrequently followed this complication. The action of pleural effusion or sterile pyothorax on the lung is much the same as that produced by air compression. The accumulation of fluid within the chest, while much slower and thus less dangerous, may continue until pulmonary collapse is complete, or until that point is reached where further encroachment upon the lung is prevented by fibroid consolidation or the restraining action of adhesions. The advent of one of these complications has often marked the beginning of the retrogression of an otherwise unfavourable infection.

The therapeutic application of intrathoracic pulmonary compression in the treatment of phthisis, while not of very recent date, is only just beginning to be appreciated.

Artificial pneumothorax is subject to the same limitations as are those diseases producing spontaneous compression within the thorax. Its successful use will, therefore, depend upon the choice of such thoracic conditions as allow of lung compression, and the maintenance of a constantly compressing force in that region which will directly bear upon the active lung process.

Clinical Application of Pneumothorax.

Unilateral cases of phthisis, relatively free from evidence of old pleuritis, recurrent pleurisy with effusion, and certain cases of lung abscess and bronchiectasis of not too long standing, offer the greatest field for its clinical application. In pulmonary tuberculosis, the range of the selection of cases

is limited only by the ability of the patient to take the treatment, and the object the operator has in view in giving it. If arrest or apparent cure of the disease is the ultimate object, then the treatment in most cases must be limited to those cases showing unilateral signs and without much fibroid tissue formation. If relief of one or more troublesome symptoms is the result sought, then any case of phthisis where the heart action is good and the pleural cavity is not obliterated may be treated.

Recurrent pleurisy with effusion may probably be shortened in its course, and the damaging action of pleural adhesions lessened by the withdrawal of the effusion and the compression of the lung lymph spaces by nitrogen.

In abscess of the lung and bronchiectasis compression should be exerted from below upwards to allow for drainage. The chronicity of such cases does not often allow of a complete arrest of the process, but frequently much can be done in modifying the symptoms.

Few incipient cases of phthisis have so far been reported as treated with pneumothorax. It would seem best for the present, on account of the complications to which the nitrogen treatment is subject, to exclude all early cases from the treatment except such as are rapidly progressing under the best of sanatorium conditions. Early cases, however, may show remarkable improvement in a few weeks, with a total loss of activity of the process in the lung as the result of artificial collapse.

The advanced cases should never be refused treatment because the collapse of the lung is thought to be impossible without the effort being made. Often the most unpromising case, as judged by all physical signs, may give very little trouble in regard to pleural capacity. The condition of the myocardium and the extent of the disease may, however, prevent the use of nitrogen. The condition of the

heart should be such as to be able to care for a readjusted pulmonary circulation, and should be strong enough not to readily dilate should pleural shock occur. While the advancing unilateral case is considered the one of choice for artificial pneumothorax, the great majority of those treated will be bilateral. If the second lung shows a limited amount of apical involvement with little activity, the effect of the treatment may be beneficial to both lungs. When the second lung shows an active apical process, even of limited extent, the effect of the treatment may be to rapidly extend this process. I have seen one such case. A basal process in the least affected side nearly always precludes the use of nitrogen compression. The increased respiratory activity thrown upon the lower lobe of the untreated lung will tend to advance the disease in this location. Occasionally, subsequent to the arrest of a case of phthisis by artificial pneumothorax, the untreated lung may take on an active process after the cessation of treatment on the other side. In such instances, if the first lung is able to carry on respiration the active lung may be compressed, provided this is done with care and the amount of compression slowly increased. Where tubercular pyothorax or effusion complicate phthisis, the compression thus exerted should not be interfered with unless the fluid is invaded by pyogenic organisms. In such cases the withdrawal of the fluid and compression of the lung with nitrogen should first be tried before an open chronic thoracic sinus is established.

The selection of the point of injection is secondary in importance only to the selection of the case to be treated. In the routine case, the lower dorsal surface of the thorax offers the best location. The majority of cases of phthisis begin at the apices and advance downward, and in these cases the pressure will first be exerted upon that portion of the lung most recently invaded, and which gives rise to the

symptoms. The respiratory activity of the diseased lung will be most rapidly retarded by pressure exerted at the base. In this region also there is apparently less danger of producing pleural shock than nearer to the root of the lung, and collapse of the lower lobe gives less distress from pressure. In cases of abscess of the lung and bronchiectasis, which most commonly occur in the lower lobe, compression can also be most favourably exerted by thoracentesis in the lower part of the chest. In the selection of the point of injection the presence of adhesions must always be considered. Where physical examination shows the lung to readily expand with inspiration, with the descent of the lung borders and a resonant note on percussion in most instances, the lung will be free. X-ray examination is, however, all-important here to prevent failure in many cases where physical signs give no indication of adhesions. No case should be undertaken without such an examination where it is possible to supplement auscultation and percussion with it. In this way frequently a few bands may be avoided which would otherwise negative all effort towards the successful production of a pneumothorax.

Methods.

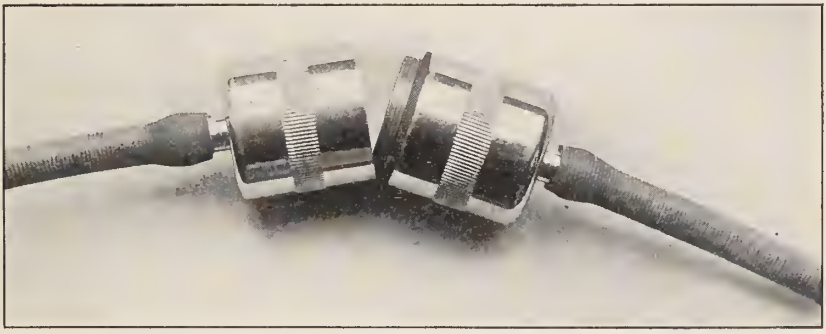
At the present time two methods are in vogue in the production of artificial pneumothorax. The incision method of Brauer has so far failed to maintain its place against the simpler puncture method of Forlanini. The latter on account of its simplicity, its greater freedom from complicating emphysema, and not being one of greater danger, is now largely in use in this country. With either method, however, constant pressure from nitrogen upon the lung should be maintained. Certain operators prefer to fairly rapidly increase the amount of nitrogen injected, and at the same time prolong the interval between injections. This

routine has the disadvantage of allowing an irregular rate of absorption of the nitrogen and an inconstant positive intrathoracic pressure. More preferable is the method of giving of small amounts of nitrogen at an interval of a day or two, thereby steadily increasing the force exerted upon the lung, and making up by the frequent reintroduction of nitrogen for an uncertain rate of absorption of the gas. The complete collapse of the lung in most cases, if of any duration, whether of phthisis or abscess of the lung, is generally impossible, but in a large proportion sufficient collapse can be obtained to give good results. It is often remarkable to see how small an area of pneumothorax will favourably influence the pulmonary condition. Often even in an advanced case, where not more than a few hundred cubic centimetres of nitrogen can be tolerated, the advance of the trouble is checked and even arrested, when apparently only a small amount of pulmonary tissue is reached. The failure to get improvement or to prevent a relapse in a given case is often due to too early allowing of re-expansion of the lung. Collapse should be maintained until all evidence of active disease has disappeared from the lung for several weeks, and then the rate of re-expansion should be a slow one. In recent pleurisy with effusion, an amount of nitrogen two-thirds in quantity of the amount of fluid withdrawn should be introduced, and this amount should be maintained within the chest. As the re-accumulating effusion diminishes in amount less and less compression will be necessary. In bronchiectasis the collapse of the lobe affected should be continued until adhesions prevent its re-expansion.

Intrathoracic Conditions.

The condition existing within the thorax, both as regards positive or negative pressure and lung movement, can be learned by the use of the manometer control. The closer

this instrument is followed the more safely will the operation of artificial pneumothorax be performed. It is never advisable to allow an inflow of nitrogen into the chest unless an excursion of $1\frac{1}{2}$ to 2 c.c. in the water column is obtained with respiration. In many cases valuable information may be obtained by the action of the manometer at the first injection. Where little excursion occurs the lung is generally in close proximity to the chest wall or tied down to it. Where a marked negative reading appears, as is commonly



Metal Cell.

the case, the lung has often been carried away from the chest wall by the retractive action of fibrous tissues. Occasionally, where a cavity has occurred near the area selected for injection, the pleural space may be entirely obliterated, and the puncturing needle may enter this cavity without causing any symptoms. In such instances the manometer readily varies without any persistent tendency to either positive or negative pressure. Nitrogen, if allowed to flow in, runs with suspicious rapidity, and this, together with the metallic taste complained of by the patient and subsequent lack of signs of a pneumothorax, will tell the operator what has occurred. If a small cell containing menthol, or some other equally

noticeable drug through which nitrogen is allowed to flow, is inserted in the apparatus prompt recognition of the passage of nitrogen out through the mouth may be obtained. Knopf speaks of the danger of entering a cavity on account of carrying its septic contents into the pleural space and chest wall. In two or three cases in which I have personally done this no subsequent infection has occurred. The danger of infecting the pleural cavity in this way is, I believe, a minor one, as the parietal and visceral pleura must be adherent in order to make this readily possible. When the needle enters the lung substance very little effect is noticeable on the manometer from respiration. If the patient moderately inflates his lungs, with the needle in the lung, no corresponding excursion occurs, and it is never safe to introduce nitrogen until it is obtained. The danger of air embolism is much overestimated, and in some four years of work with this method I have never seen a case. Furthermore, experiments on animals would seem to show that only exceptionally can it be brought about even when the effort is made to produce it. The amount of nitrogen which may be given to a patient at one time will depend largely upon the amount of positive pressure shown by the manometer, accompanied by definite symptoms on the part of the patient. Where the needle enters a small pleural pocket, and this is filled with nitrogen, pleural pain and positive pressure will rapidly occur. In those cases where adhesions are old and firm no symptoms may follow even vigorous efforts to introduce nitrogen, as shown by a marked positive pressure. If, however, the inflammatory condition of the pleura is of shorter duration positive pressure may be succeeded by atmospheric or even negative pressure as the adhesions give way under the advancing gas, and it may fail to again rise even with large amounts of nitrogen. It is well not to force such a case too rapidly, especially at the first injection.

Perseverance under those conditions which at first may seem hopeless will in time allow of an extensive pneumothorax and often good results.

The danger of rupturing the lung and producing pleural hæmorrhage by the giving way of adhesions is a very slight one, and as far as I am aware none have been reported. When the treatment is well under way, at the close of each injection the manometer should register a slight amount of positive pressure, thus showing that a certain amount of force is exerted upon the lung. Subsequent injections should be large enough, or given often enough, so that at least atmospheric pressure is maintained in the chest. In this way alone can the collapse and re-expansion of the lung continually recurring be prevented.

The changes brought about following the introduction of nitrogen are not only demonstrable by physical signs, but also pathologically Clinically, the use of the X-ray has in many instances served to give a much clearer idea of the actual conditions within the chest than could be obtained in any other way. Not only can the area of consolidation with cavity formation be marked out, but the location and density of adhesions traced. The position of a body of nitrogen with its effect upon the lung obtained by this means is of utmost value to the clinician. Often the plate will show a limited body of gas markedly compressing that portion of the lung in contact with it. All possible locations and singular situations in the chest are at times taken by a pneumothorax. The diaphragm may be greatly depressed by the gas, or it may extend in a thin sheet over a considerable portion of the lung showing no great depth at any one point, or a series of pockets of varying size and shape separated by adhering bands may occur. Now and then where the lung is united to the chest wall by many adhesions as the gas burrows in among them the lung may

collapse before it, leaving numerous outstanding strands extending through the air cavity. I have seen one patient in whom the tension on pleuro-pericardial adhesions was so great following a pneumothorax as to produce great cardiac embarrassment and necessitate its withdrawal. Physical examination will demonstrate changes according to the extent of the pneumothorax and the existing thoracic conditions. The loss of pleural, and at times of pulmonary, râles in the area of compression is quite striking. In the incipient lesion one injection alone may permanently obliterate all pulmonary moisture. This is produced by the limitation of air movement in the compressed area and the collapse of the alveoli.

The signs of amphoric respiration and a "cracked pot" note on percussion in cases of pulmonary abscess or phthisis may cease to be demonstrable after compression. Extensive modification of the physical signs in the compensating lung is often noticeable. These changes, as has been pointed out, are due to the enforced activity of the second lung, and when a pneumothorax of any extent is established they nearly always occur. The demonstration of an artificial pneumothorax may at times be difficult. Often a modified tympanitic note on percussion is all that can be obtained to locate the presence of nitrogen even where a large quantity has been given. The peculiarities of distribution of nitrogen in the chest, as shown by the X-rays, serve to explain its cause. A diaphragmatic pneumothorax, or one film-like in type, gives little in the way of characteristic signs. If a body of gas is under tension few characteristic signs are present and the coin sign is often absent.

The pathological anatomy of the collapsed lung has been studied by Graetz, Saugman and Forianini. They all have noted the increased tendency to fibrosis, and in certain situations where collapse has been complete, alveoli, and even

bronchi, may be almost overgrown by fibrous tissue. The effect on the lymphatic and pulmonary circulation of compression is prompt and lasting. As the lung is more and more compressed the circulating blood diminishes proportionally and the lymphatics that are not obliterated by fibrous tissue dilate.

Perhaps the most noteworthy observation is the fact that although alveoli may be firmly pressed together for months there is little tendency to adhesion of their walls, and if re-expansion occurs they readily resume their function. This is of great importance, as in many instances where the lungs are allowed to re-expand it is an added protection to them to have aeration re-established. Pathologically, the end sought is obtained by the infiltration of tubercular tissue by connective tissue with its accompanying permanent restrictive action.

Complications.

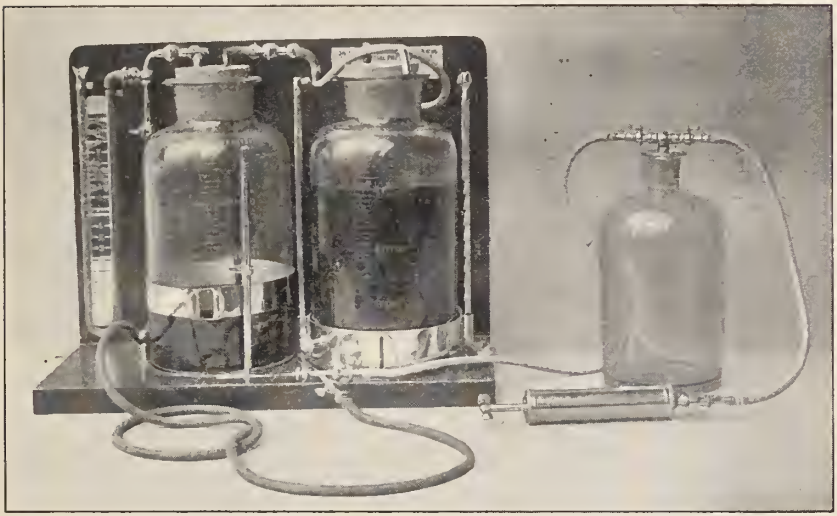
Much has been said in regard to complications which may follow the production of artificial pneumothorax. The most important one is pleural shock from irritation of the pleura upon the introduction of the aspiring needle. In spite of all precautions, in endeavouring to cocainize the pleural surface this may occur. The one fatality that I have seen was due to this cause. The symptoms are a sensation of pressure in the chest or in the vicinity of the heart, cyanosis, shallow respiration of the gasping type, with a very rapid faint heart-beat and dilatation of the pupils. It comes on without warning, and may pass off again in a few minutes, with retraction of pupils, improvement in the pulse and deepening of respiration. Sometimes before this stage occurs the colour of the lips will be alternately blue and very pale. Some patients are apparently much more susceptible than others, and the introduction of the needle in the vicinity

of the root of the lung is more likely to bring on the shock. The one case in which I have seen this complication survived the first attack and some months later succumbed to a second, as it was followed by cardiac dilatation. I consider this complication the most important one among those associated with this operation.

Pulmonary embolus is very infrequent, and by controlling the injection of nitrogen with manometer readings this can almost always be avoided. It has occurred, however, even with so careful an operator as Brauer, and may leave a hemiplegia from the localization of nitrogen in the brain. Pulmonary hæmorrhage from injury to the lung is also infrequent, even where a violent spasm of coughing may drive the lung against the needle-point. By far the most common condition following pleural inflation is the accumulation of an effusion. This generally comes on after several inoculations and may at first be so small in amount that even the X-ray will fail to detect it unless the patient is in the upright position when examined. Where injections are very frequent and small amounts of nitrogen are given at a time it is more likely to occur than where the interval is of some duration. Examination of the fluid will occasionally show the tubercle bacillus to be present. Now and then, either from lack of careful technique or from extension of pyogenic organisms through the lung substance, the fluid may become contaminated and pyothorax become established. This is almost always a serious condition. Where the fluid accumulates in any amount it seems best to withdraw it and to keep the lung well compressed with nitrogen. In my own work I have seen very few effusions of any amount. Occasionally spontaneous pneumothorax may supplement an artificial one by the giving way of the lung substance. This may or may not be of serious import according to conditions present. If tension upon a thin-walled cavity results in breaking it, the contents may escape into the pleural cavity

with resulting pyothorax. In most cases fibrous tissue prevents this. Where the Brauer method is used for the initial introduction of the gas, not infrequently the wound shows a low grade of sepsis. I have never seen an infection of the chest wall following the puncture method.

Subcutaneous emphysema commonly occurs where nitrogen is under considerable tension in the chest. Its presence is troublesome but not serious, unless it perforates



Pneumothorax Apparatus connected for Aspiration and Inflation.

the costal pleura and invades the mediastinum. In such a case the symptoms give cause for anxiety.

In pulmonary tuberculosis complicated by a tubercular condition of the kidney, the use of artificial pneumothorax is generally precluded on account of the danger of suppression of urine. If it is used at all it should be done with great care.

The rapidity with which the lung may collapse is of importance, in order that the load thrown upon the right ventricle may not be too suddenly increased. The ready

inflow of nitrogen into the thorax, with the rapid increase in the signs of pneumothorax and the failure of the manometer to indicate any degree of positive pressure, all indicate that the lung is steadily retreating. I have seen one case of temporary right-sided cardiac dilation from too rapid collapse of the lung. It is maintained that collapse, and later re-expansion of compressed pulmonary tissue, sooner or later tends to spread the disease through such uninvolved lung as a result of its lowered vitality following the treatment. This is merely one more argument for as complete collapse of the lung as possible and its maintenance in such condition until all activity has ceased.

In a certain number of cases that I have followed, in which a pneumothorax was produced about three years ago, while the general condition was greatly improved and the disease brought to a standstill, although all activity had not disappeared, the subsequent history has shown an extension of the disease into the re-expanded portion of the lung. The protection accorded the untreated side through an abundance of blood and air has prevented any development of active disease there.

In pleurisy with effusion after the withdrawal of the fluid, the tendency of the compressed lung is to slowly re-expand until it is recompressed by a re-accumulation of serum stimulated by the presence of the tubercle bacillus. The injection of nitrogen will maintain the lung at the point to which it has been carried by the effusion, and while it will not prevent the recurrence of the fluid it will help to diminish its quantity, and therefore aid in a more rapid recovery. In cases of lung abscess or bronchiectasis, unless the nitrogen injected is restricted by adhesions, the lung substance about such a process will be driven in upon the infiltrated area, and even the cavity itself may be occasionally compressed into a smaller compass.

Not infrequently, however, especially in the older pro-

cesses on account of the extensive inflammatory reaction, little can be accomplished.

Results.

Two years ago, in conjunction with Dr. Robinson, I reported a series of twenty-eight cases treated by artificial pneumothorax. Of this number some eight cases had either shown an entire arrest of an active process or were approaching that condition. The other twenty cases had secured relief of symptoms to a greater or less degree. Of these arrested cases six are in excellent condition to-day. One has died through no fault, however, of the method, but rather on account of the ignorance of the patient, and one has within the last year shown an extension of the old process into the previously collapsed area of the lung. These few cases may, therefore, be added to an already growing number of cases of pulmonary tuberculosis that have not only been arrested temporarily, but also probably permanently by artificial pneumothorax.

During the past four years I have been enabled to treat a fairly large number of cases of pulmonary tuberculosis by means of nitrogen compression. Some of these have been under treatment primarily for the relief of one or more symptoms, such as persistent cough or hæmoptysis. In the great majority, however, the object of the treatment has been to arrest an advanced active process. In the larger number of cases reduction in temperature, relief of cough, diminution of expectoration, and an increase of body weight have occurred. In several cases where the character of the disease was of a chronic toxæmia with an advanced pulmonary lesion, the results of the compression of a portion of the active lung has markedly diminished toxic absorption. Improvement in circulation, gain in weight, and increase of strength followed even where the extent of pneumothorax was limited. In one case of pyothorax, secondary to an

advanced tubercular process of the lung, in which the pus was withdrawn and the fluid replaced by nitrogen, no results were obtained on account of the dense, unyielding condition of the consolidated lung. In cases of mixed pulmonary infection, results have been fairly good only where collapse of the lung has been extensive.

The compression of a portion of the lung tends merely to diminish the amount of sputum and reduce temperature. The action of pyogenic organisms is not checked, and lung destruction slowly advances.

In unilateral cases of active tuberculosis, not only relief of symptoms, but arrest of the disease has been obtained. The time required to accomplish this has varied with the extent and activity of the disease treated. The tendency in most instances is to lengthen the interval of injection too rapidly, and even stop them altogether before permanent arrest is assured. With artificial pneumothorax, as with other methods of treatment of tuberculosis, relapses occur, and a renewal of the treatment may be necessary. Thus, after allowing the lung to re-expand and to begin the formation of new adhesions, it may be quite difficult, and may require considerable patience to again recollapse it. Nitrogen may have to be forced in under considerable water pressure in order to separate the pleural surfaces, even in those cases where the first series of treatments have uniformly been carried out without difficulty. After establishing a moderate pneumothorax, the lung generally collapses more rapidly from this point onward than with the first few injections. The same difficulty also arises in treating those patients who are not systematic in following up the treatment, and allow weeks to intervene between injections.

In a few cases of pulmonary tuberculosis where the pulmonary process has been extensive in one side and inactive in the other during the series of injections, renewed

activity may occur at the margin of the old inactive process. In such cases continued compression merely aggravates the focus of the disease, and it is often wisest to desist entirely from the treatment, or at most to maintain only a moderate pneumothorax. Occasionally it has been possible to arrest the disease in one half of the chest, and later, after allowing this lung to re-expand, to compress the second which has shown evidence of activity. A few such cases have occurred among those reported by German observers. In such instances as I have seen, in which the second lung has become actively involved, the condition of the patient was not such as to allow of any extension of the method.

It has been the experience of some operators, after having established a pneumothorax in one side, to have pulmonary hæmorrhage occur from the untreated lung. This has been due to the increase of pulmonary circulation through a portion of lung previously damaged by disease which has given way under increased tension. Such difficulties are, fortunately, infrequently met with, and where the amount of bleeding is at all extensive may preclude any further use of nitrogen.

Sometimes it may be thought desirable to remove a certain portion of the amount of nitrogen injected into the chest on account of cardiac symptoms following a well-marked collapse of the lung. Where no more than atmospheric pressure has been produced within the chest cavity suction may be necessary to remove any appreciable quantity. This, however, is rarely necessary.

After some experience with the method of artificial pneumothorax, one is struck with the relatively small number of those afflicted with pulmonary disease that are really suitable cases for this treatment. Well-marked or advanced strictly unilateral cases are not especially common even in large clinics for pulmonary tuberculosis, and some observers

believe that such cases are always bilateral. If this type of case is strictly adhered to, the results obtained where the method is carried out conscientiously will be uniformly good, and the percentage of patients markedly improved or even arrested will be as large as 40 or 50 per cent.

If, on the other hand, as is the tendency with most men, almost any otherwise hopeless case in which satisfactory compression can be obtained is given a chance of obtaining relief, at least from persistent symptoms, the results will not be brilliant, and I believe not more than 5 or 10 per cent. possibly of such cases will be arrested.

Artificial pneumothorax will be of real aid in the chronically recurring tubercular pleural effusion, and in some cases of bronchiectasis and pulmonary abscess, in which the inflammatory conditions to be met are not too extensive. But even if this group of diseases may be benefited by the use of this method, its field in the treatment of diseases of the chest will still, I believe, be relatively a narrow one. It is most applicable where the patient is under the supervision of hospital or sanatorium management, and has failed to do well with the older and well-established methods.

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NOTE.—For the discussion of this paper see pages following paper by Drs. Barnes and Fulton.

A REPORT OF SEVENTEEN CASES OF PULMONARY TUBERCULOSIS TREATED BY ARTIFICIAL PNEUMOTHORAX.

By HARRY LEE BARNES, M.D., and FRANK TAYLOR FULTON, M.D.

RHODE ISLAND.

The seventeen cases included in this report were treated in the Rhode Island State Sanatorium since January, 1912. The primary injections in the first two cases were made by Dr. Cleaveland Floyd, to whose kindness in assisting us at the outset we wish to make acknowledgment. Only nitrogen gas was used, the first few amounts being obtained from a chemist, and the remainder being made in the sanatorium by removing oxygen from air by means of pyrogallic acid and lye. An improvised apparatus and manometer was used until a Robinson-Floyd apparatus was obtained. The Forlanini method of injection was followed in all cases. Anæsthesia was at first obtained by $\frac{1}{10}$ per cent. solution of cocaine, but as one alarming syncope was believed to have been caused by it $\frac{1}{2}$ per cent. solution of novocain was substituted to our satisfaction. Ten men and seven women were treated, and only far advanced, progressive, or unimproved cases were selected. No X-ray being available, we were forced to rely on physical examination. In fourteen, or 82 per cent. of the cases, gas was readily introduced. The literature has been so well reviewed by Robinson and Floyd,

Hamman and Sloane, Balboni, and others, that no such review will be made by us.

Case 1466, female, aged 25, duration of disease eleven months, fever continuous for eight months in sanatorium, except occasional intervals for a few days. Dulness and medium moist râles heard over left upper lobe front and behind, and the apex of lower lobe posteriorly; subcrepitant râles from right apex down to first rib, and a few over apex posteriorly. Cough and expectoration increasing and signs slowly progressing in left lung. Gas injections were made into the left pleural cavity.

	Cubic centimetres
January 2, 1912	500
January 12	1,000
January 31	1,200
February 12	1,400
March 4	1,700
April 10	1,300
May 21	325
July 6	75
October 15	150
December 20	210
January 29, 1913	240
February 14	200
March 19	350

On May 21 the pressure became positive, and has been so ever since, the manometer showing 2 to 7 cm. of water-pressure.

Result.

Following all injections the same phenomena were noted. The cough and expectoration were markedly increased for from twelve to twenty-four hours, followed by almost complete cessation of cough. The sputum was reduced from 65 to 30 c.c. by the first injection. Tubercle bacilli, which had never been absent during the eight months, left the sputum after three months' treatment, and have not returned for the year that has elapsed. There is only about 10 c.c. of sputum per day. There is still a slight return of the cough about a month after a gas injection, which persists until another injection is given. Patient works four and a half hours in addition to walking exercise.

Case 1570.—Male, aged 25, duration eight months, of which five have been spent continuously in bed in the sanatorium, with afternoon temperature usually between 100° to 101° F. The left lung infiltrated from apex to base, signs of a cavity appeared in the lower lobe of the left lung posteriorly about six weeks after admission, and a slight amount of fluid at the left base was absorbed during the following months. On the right side slight dulness and harsh breathing extending from the apex down to the second rib with a few râles. Evidently a hopeless case. Gas injections into the left pleural cavity.

	Cubic centimetres
January 2, 1912	500
January 12	1,000
January 31	1,200
February 12	1,500
March 4	1,600
April 10	1,800
March 23	1,800
April 26	350
June 7	525

The first seven injections were uneventful, but after 350 c.c. of gas was introduced on the eighth injection he collapsed, and was unconscious for over forty minutes, the pulse not being perceptible at first. The patient made a good recovery from the collapse, no permanent harm resulted, and he had one treatment afterwards. Cases of collapse occur occasionally after the use of cocaine, even though the solution is dilute and the dose small. One-tenth of 1 per cent. cocaine was used in this case, the dose being about $\frac{1}{18}$ gr., and we have felt that the collapse was due to this drug, possibly because of the needle entering a small vein. The pressure was always negative in this case.

Result.

After the first injection the afternoon fever, which had ranged from 100° to 101° F., fell to an average of about 99·5° F., being frequently normal. The patient gained $2\frac{3}{4}$ lb., and the sputum, which had amounted to from 70 to 180 c.c., dropped to 25 c.c., the cough showing corresponding improvement. June 8, 1912, the patient began a series of eight hæmorrhages in sixteen days, totalling 27 oz. of blood, which clearly came from the right lung, and as it was not considered feasible to compress the right lung the treatment was abandoned. The patient died fourteen months after pneumothorax was established.

Case 1579.—Male, aged 24; duration of disease five months previous to admission. After seven months in the Sanatorium, during which he had slight fever 99° to 100° F. about a third of the time with four attacks of blood-spitting, he had another hæmoptysis, during which he lost 7 oz., the fever being 101° to 103° F. Dulness and broncho-vesicular breathing over upper lobe, and large, moist râles over the whole right lung. The upper lobe of left lung also infiltrated. The first gas injection of 650 c.c. into right pleural cavity was given during the hæmorrhage on April 10. The hæmorrhage stopped, and did not recur during the remainder of his stay of three months. Subsequent injections were:—

	Cubic centimetres
May 1, 1912	... 925
May 21	... 1,200
June 19	... 1,450

RESULT.

Temperature came to normal five days after first injection, remained normal a week, and then recurred. After fifth and last injection temperature remained normal, until patient left institution to return to Portugal. Sputum reduced from 50 to 25 c.c., but no improvement in general condition, his weight gradually dropping until he had lost 6 lb.

Case 1577.—Male, aged 18; duration thirteen months, of which eight had been spent in the institution, and during which he had had two small hæmorrhages and streaked sputum on two other occasions, the last being one month before the first gas injection. He had gained well on first entering, but for three months previous to gas treatment he had been slowly losing weight and feeling badly. Temperature usually normal. Over upper lobe of right lung was slight dulness, broncho-vesicular breathing, and moderate number of moist râles. Middle lobe also shows signs of commencing disease. Two small patches of infiltration in the left upper lobe. Gas injections in right pleural cavity given without difficulty or discomfort, the pressure being strongly negative.

	Cubic centimetres
May 1, 1912	... 650
May 21	... 1,000

On June 15 and 17 the patient had slight hæmorrhage of 3 and 2 oz. respectively. On June 16 patient had slight chill and vomiting, temperature rising to 102° F. On the 18th the temperature fell to 99° F., and patient's stomach symptoms disappeared. On the morning of June 19, 1,225 c.c. were injected without difficulty or discomfort. June 20, marked cyanosis and dyspnœa. An unsuccessful effort was made to remove the gas, the negative pressure being too strong. June 22, cyanosis better, but dyspnœa continued. Scattered areas of broncho-vesicular breathing and râles in previously healthy areas of both lungs. Amount of sputum doubled. The signs of broncho-pneumonia soon became typical, thus accounting for cyanosis and dyspnœa. Patient emaciated, steadily lost strength, and died July 11, three weeks and two days after last injection.

Case 1648.—Male, aged 21; duration twenty-two months, of which nine months had been spent in the Sanatorium. Patient had gained 24 lb. in weight, but had had six hæmorrhages and many slight temperature attacks. Right lung, slight infiltration, with few râles above the third rib anteriorly, and only at apex posteriorly. Left upper lobe, moderate dulness, broncho-vesicular breathing, and large, moist râles. Upper part of lower lobe also involved, and lesion apparently extending. Gas injections into the left pleural cavity.

	Cubic centimetres
May 21, 1912	... 650
June 7	... 1,200

Patient did not improve as fast as he thought he should, refused further treatment, was discharged August 26, and has been working ever since.

Result.

The gas given was insufficient in amount to expect much result, and there was no striking change in his condition. Patient has had no more hæmorrhages, but whether this can be credited to the pneumothorax is most uncertain.

Case 1716.—Female, aged 27; duration thirty-two months, of which three months were spent in the Sanatorium. The first three months in the institution she had neither gained

nor lost weight, her temperature occasionally going to 100° F. Left lung completely involved with consolidation in the upper part and cavity signs below the clavicle. Many large moist râles all over the lung. Harsh breathing and few subcrepitant râles above the second rib on the right side. April 22, 1912, patient had chills. Her temperature went to 102° F., and kept high almost continuously, with an occasional morning drop to normal. Patient had refused pneumothorax. May 9 signs of another cavity appeared in the upper part of the left lower lobe posteriorly. Gas injections into the left pleural cavity, June 7, 1912, 450 c.c. Patient's temperature continued to go up occasionally, but had a two-day normal interval. June 25, 450 c.c. produced positive pressure. Temperature made a decided drop to an afternoon average of about 99·4° F., with marked relief to the patient. Sputum continued about the same amount.

	Cubic centimetres
July 6, 1912	450
July 14	400
July 27	200

all producing positive pressure and slight dyspnœa. Patient's sputum continued the same in amount. She steadily lost weight and refused more injections, but the temperature continued nearly normal until August 28, when evening temperature again ran from 100° to 102° F., and patient was discharged.

Result.

Striking reduction of temperature followed pneumothorax, and continued as long as positive pressure was kept up. Patient was obviously too far advanced to be saved.

Case 1785.—Male, aged 27; duration thirty months, the last month in the Sanatorium. Had previously had over a year of treatment in two different sanatoria, besides treatment in several health resorts. Temperature nearly normal. Hæmorrhage of 1 oz. about a month before injection. Has lost slightly in weight every week since admission. Slight dulness, broncho-vesicular breathing all over right lung, with many large, moist râles. Slight broncho-vesicular breathing, occasional subcrepitant râles in upper lobe and upper

part of lower lobe of left lung. Patient was discouraged by his continued failure in different health resorts and sanatoria. Injection of gas, June 7, 700 c.c. into the right pleural cavity. June 8, two hæmorrhages of 3 oz. each. June 10, several hæmorrhages in last two days, totalling 20 oz. of blood. June 13, 1,225 c.c. of gas injected during a hæmorrhage. Temperature, 101·8° F. June 18, three hæmorrhages, giving a total of 5 oz. June 19, 525 c.c. of gas injected. Hæmorrhages ceased, but temperature continued 99° to 100° F., with occasional rises to 101° F. All injections had been stopped on negative pressure, and were uneventful. July 16, 100 c.c. gas injected. Injection discontinued because of pain. July 23, patient developed diarrhœa, accompanied by abdominal tenderness, which was believed to be due to tuberculous enteritis. Patient left the institution, and died five months after the first injection.

Case 1645.—Male, aged 30; duration sixteen months, of which ten had been spent in the Sanatorium. Had gained 7 lb. in weight in the first four months, and then began to have spells of temperature. Both lungs were markedly involved, the left being decidedly the worse, with signs of cavity formation below the clavicle, the right lung, however, having slight broncho-vesicular breathing and râles through the upper lobe, and a few râles in the upper part of the lower lobe posteriorly. Gas injections into the left pleural cavity.

	Cubic centimetres
June 7, 1912	700
June 25	200
July 6	100

Except the first time, only small amounts could be injected, as the manometer registered positive, and the sense of pressure was uncomfortable. Four different places were tried. Sufficient gas could not be introduced to expect any result, and there was none. The patient lived eight months after the treatment was instituted.

Case 1783.—Male, aged 36; duration seven months, including one in the Sanatorium. Fever continuous from 100° to 105° F., with prostration. Left lung moderate, dulness very marked, bronchial breathing, increased voice conduction,

and moist râles from top to base. Upper lobe of right lung infiltrated. Gas injections into the left pleural cavity. June 7, 125 c.c. caused positive pressure and pain. June 25, 200 c.c. caused positive pressure and pain. Four points of injection were tried.

RESULT.

Failure to inject sufficient gas. Patient died about two months after the first injection.

Case 1431.—Female, aged 35; duration seventeen months, of which fourteen were passed in the Sanatorium. Patient was in bed with fever for nearly thirty weeks after admission, the fever running between 103° and 105° F. for nine weeks, with much pleuritic pain in the left side. Patient had been in bed with many spells of temperature since. She had had six small hæmorrhages and streaked sputum on many occasions. Had been in bed two weeks with temperature between 99° and 100° F. at the time of first injection. Left lung has a few subcrepitant râles in the lower lobe, and patient has had almost constant pleuritic pain in this area. Right lung also has subcrepitant râles over upper lobe and apex of lower lobe posteriorly. Gas injections into the left pleural cavity.

	Cubic centimetres
June 25, 1912	650
July 6	900
July 16	750
July 27	800
August 27	450
September 11	450
September 27	500
October 15	350
December 20	725
January 29, 1913	785
February 6	750
March 19	550

The first injection caused enough dyspnœa to be rather uncomfortable, and this symptom was noted to a less degree on two other injections. Positive pressure varying from a slight amount up to 3 cm. of water was produced at the end of the last four injections.

RESULT.

Patient is much improved, but is still far from being well. Her sputum is about the same in amount, and usually con-

tains bacilli. She had a hæmorrhage on the 15th and also on October 17, and streaked sputum on two other occasions, but she has had almost complete relief from the pleurisy in the left side, and although she quite occasionally has a temperature of 99·4° to 99·6° F. in the afternoon, she has not been in bed with a temperature regarded as due to tuberculosis since the second injection. Is still in the Sanatorium.

Case 1597.—Female, aged 15; duration fifteen months, of which nine months had been spent in the Sanatorium. Nearly all of the first eight weeks after admission were spent in bed. The fever constantly tended to recur, and patient had been in bed or on her bed over half the time. She had, however, gained 14 lb. between October, 1911, and March, 1912. From May 14 to June 25 her cough increased, she began to have slight temperature up to 100° F. more frequently, and slowly lost weight—5 lb. in all. There was considerable consolidation, with moist râles in the upper lobe of the right lung, with cavity signs near the apex. The upper part of the lower lobe was involved posteriorly. Signs in the left lung covered about as much area, but there were no signs of excavation. Gas injections into the right pleural cavity.

	Cubic centimetres
June 25, 1912	500
July 6	750
July 16	700
July 27	500
August 27	500
September 11	500
September 27	500
October 15	500
November 7	500
December 20	825

The last injection was the only one in which a positive pressure was recorded. After the third injection the temperature fell to normal, and remained there for several months, except for an occasional 99·4° F. once in two or three weeks. She regained the 5 lb. which she had lost and coughed less, but the amount of sputum kept the same, tubercle bacilli being still present. The improvement only lasted about five months, as temperature and cough began to increase after the last injection, which was about a month before discharge.

Case 1788.—Male, aged 26; duration of disease thirteen months, one of which had been spent in the Sanatorium constantly in bed, with fever from 99·5° to 101° F. almost, never normal. Marked weakness. Upper lobe of left lung had consolidation and excavation, with many moist râles. Upper part of lower lobe also had moist râles. Upper lobe of right lung badly infiltrated. Hæmorrhage or streaked sputum on twelve days of the first month. Gas injections into the left pleural cavity.

	Cubic centimetres
June 25, 1912 ...	... 700
July 16 ...	... 1,000 resulting in positive pressure.

Result.

The patient continued to fail steadily, and died July 30, 1912, apparently uninfluenced by the treatment.

Case 1800.—Female, aged 29; duration eight months, of which three had been spent in the Sanatorium. Was a third of the time in bed with recurring temperature attacks. Had gained but 5 lb. during this time. Upper lobe of the left lung much infiltrated, also apex of lower lobe. Upper half of right upper lobe infiltrated. Gas injections into the left pleural cavity.

	Cubic centimetres
August 27, 1912	500
September 11	800
September 27	850
October 15	800
November 7	800

Result.

Pleuritic pain and temperature followed the first injection for four days, upon which the temperature fell to normal, and remained there for five months, during which time the patient gained 8¼ lb. Patient declined to take further treatment, and temperature is commencing to return. The sputum was not reduced in amount, but since the gas has been absorbed the amount had doubled.

Case 1752.—Female, aged 14; duration seventeen months, of which five had been spent in the Sanatorium. In bed continuously since admission, with temperature of from 99·5° to 102° F., was growing weaker, but held her own in weight.

Left lung, upper lobe consolidated, with signs of large cavity at the second interspace and much moisture.

Upper part of the lower lobe infiltrated, with signs of moisture. Signs in right lung cover much the same area, but signs of consolidation and excavation are absent. Only the bases of the lungs were free from signs. A desperately sick case, in which the advisability of giving the treatment was very doubtful. Gas injections into the left pleural cavity.

	Cubic centimetres
August 27, 1912	100
September 11	200
September 27	350
October 15	350
December 20	400

Patient was subject to attacks of syncope, and had a slight attack during first injection, which completely passed away in five minutes.

Result.

Following the second injection the temperature fell to normal, and remained there for over four months, or during the remainder of the treatment. The patient gained strength, was up and about, and the sputum fell to about one-half its former amount. Declined to continue treatment.

Case 1846.—Female, aged 21; duration of disease thirty-one months, of which one had been spent in the Sanatorium. Fever had run between 100° and 103° F. for a month previous to the first treatment, during which cavity signs had commenced to appear at the right apex. Right upper lobe partially consolidated with pronounced moisture. Middle lobe and apex of lower lobe infiltrated. Râles from left apex down to the second rib anteriorly. Gas injections into the right pleural cavity.

	Cubic centimetres
August 27, 1912	350
September 11	750
September 27	750
October 15	800
November 7	600
December 20	50
January 29, 1913	190
February 14	300

Manometer showed positive pressure at the close of all

injections. After the first two only slightly positive pressure could be tolerated.

Result.

Temperature reduced to below 100° F. after the second injection, and almost to normal after the fourth injection. Marked general improvement. Patient is up and about, and has gained 13 lb. since the first treatment. Is still in the Sanatorium.

Case 1995.—Male, aged 20; duration forty-two months; a chronic relapsing patient who had been in other institutions, and who wished to try pneumothorax. Temperature normal. The right lung is infiltrated from apex to base. Subcrepitant râles heard all over. Subcrepitant râles also heard at left apex. Gas injections into right pleural cavity.

	Cubic centimetres
January 29, 1913	100
February 6	500
February 14	25

Gas introduced the first time after one failure, and the last time after two failures. They were accompanied by considerable pain at the site of injection, and the patient left the institution to obtain a new treatment of another kind. No result.

Summary of Seventeen Cases.

Of the Condition of Patients.—All had bilateral disease. All tubercle bacilli in the sputum. Nine were far advanced and progressing. Four were moderately advanced and progressing. Twelve were chronic fever cases. Six had cavities. Sixteen had failed to gain in the Sanatorium after an average duration of six months.

Of the Results.—Three could not be given gas enough for a fair test. Seven had their temperatures reduced to normal. Five had their afternoon fever reduced to an average of 99·5° F. Nine gained weight. Eight abandoned treatment after marked improvement. Two developed hæmoptysis in the opposite lung. One developed bilateral

tuberculous broncho-pneumonia. Seven died with an average duration of life of 5·2 months after treatment was instituted. One most discouraging fever case has had arrest of the disease with absence of bacilli for over a year.

While seventeen cases are obviously too few to form definite conclusions, it is our impression that pneumothorax prolonged the life of 12, or 70 per cent. of these far advanced and progressive cases. We would expect much better results in unilateral cases and in those less far advanced. Most moderately advanced progressive cases in which high or moderate fever is not controlled within a month or two by rest in bed, and in which it is clear in which lung the disease is active, are entitled to a prompt trial of this method, especially as the chance of recovery in these cases is slight when treated by ordinary methods.

DISCUSSION.

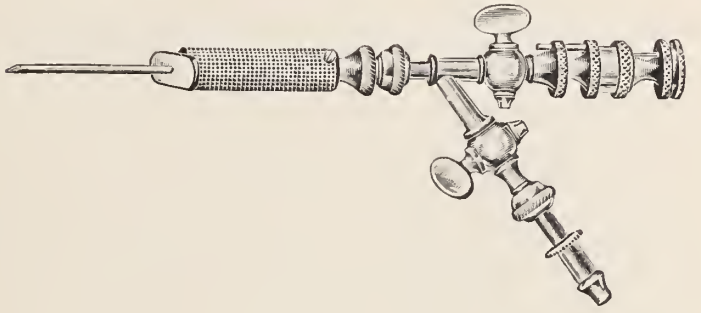
Dr. Otis : I have had occasion recently to observe the operation of artificial pneumothorax, if it may be dignified by the name of operation, in Europe, where it has become firmly established as a valuable and added resource to our means of controlling tuberculosis; more especially in cases which have not responded to the usual methods of treatment, cases which seem to be otherwise doomed. It is in these doomed cases, where the disease is unilateral, or chiefly so, that it has, in my opinion, its chief application, and the voluminous literature upon the subject now at hand has demonstrated its value in such cases and shown many strikingly successful results. There seems to be a difference of opinion as to the cause of the fatal accidents, fortunately few, as a direct result of the gas injections. Whether or not so-called "pleurax shock" is really that, or is not, in fact, gas embolism, seems to be a debatable question with many authorities. However this may be, gas embolism is the one danger most to be feared and guarded against. In order to minimize this danger of gas embolism, Professor Deneke, of St. George's Hospital, Hamburg, has devised an apparatus which, in my opinion, is the best of the numerous ones I saw in Europe. In brief, it is made double, upon one side of an upright standard is the complete apparatus for the injection of nitrogen gas, and on the other side is a replica of the same for the injection of oxygen gas, Professor Deneke's idea being that greater safety is insured if on the first

injection oxygen gas is used, and if by any chance the needle may have entered a vessel, the oxygen gas, on account of its rapid absorbability, will be much less likely to produce gas embolism. By simply turning a stop-cock nitrogen gas can be substituted for the oxygen gas. At my suggestion this instrument of Professor Deneke has been imported into this country. In every case it is essential that a reliable X-ray picture should be taken before beginning the injection to determine the condition of the lung, and existence or not of pleuritic adhesions, and subsequently to determine how far the lung has been collapsed. So far as my observations went, the Forlanini puncture method was the one generally adopted, and not the Brauer open method.

Dr. GRIFFIN: We have had a series of cases about as large a number as Dr. Barnes has reported, and we feel every confidence that the majority have been much benefited by the treatment, and in perhaps two or three instances we feel that the patients have been brought back from a very perilous position. The most striking case was not, strictly speaking, advanced. The patient had slight signs of tuberculosis of the left lung, accompanied by a constant temperature, and it was because of this constant temperature that pneumothorax was finally induced. Following the first one or two injections the temperature promptly became normal. Only some twelve to fifteen injections were given, and now for a matter of seven or eight months she has had no nitrogen. The patient looks and feels perfectly well, and walks four or five miles a day. The nitrogen has been apparently all absorbed, because three or four weeks ago when I listened at the chest I could get a distinct friction rub at the very base. This case will not require any further injections if she does as well as she promises to do at present. I think it is an important question to determine the indications for renewed injections in any case under treatment. What guides have we to go by? It seems to me that the cough is a very decided indication. If it commences to increase, it is time to consider another injection; if the amount of sputum commences to increase, that is another sign, and if the needle upon being introduced shows a marked negative pressure, that is perhaps the most sure and definite sign.

Dr. J. ALEXANDER MILLER (New York City): I am extremely interested, as we all are, of course, in this method, and the problems which have confronted me are perhaps a little different from those reported. The experience which I have had in twenty-five cases convinces me that the percentage of far-advanced cases in which you can get very marked results, as Dr. Floyd has said, is limited certainly to not more than 10 per cent. But we have had very good results in New York in cases not advanced, but where they had very marked hæmoptysis, that frequently being the first or only symptom, and I have treated several such cases with very remarkable success. In three of them they had been bleeding from a pint to a quart a day,

and with rising temperature, and with no evidence of ceasing of hæmorrhage. After the injection of gas the hæmorrhage stopped, and prompt reduction of fever resulted. We have in such cases, I believe, an indication for prompt pneumothorax treatment. Whether it should be continued or not, after the hæmorrhage is controlled, should be decided for each individual case. I think in the chronic, advanced cases you get hung up and are afraid to stop, and yet really feel that it is not justifiable to keep on. We expect in the future to use it in more acute cases with hæmorrhage, and also in the acute febrile cases with limited lesions. One of my cases may be of interest as a rather unusual form of shock occurred. It was a hæmorrhage case and was bleeding at time of operation. Morphine had been administered previously in full doses. Careful local anæsthesia down to the pleura was employed by means of novocain; 900 c.c. of the nitrogen gas was given at the first injection, and there



was a minus 1 pressure, with absolutely no discomfort to the patient. Suddenly, just as I was withdrawing the needle the patient turned pale, the pupils dilated, the patient broke into a profuse perspiration, and his heart action became weak, he was faint, but did not lose consciousness. The particularly interesting thing about it is that this happened just at the end of about fifteen minutes' injection of 900 c.c. of gas, and the pulse instead of getting rapid and irregular became slower and slower until it reached a minimum of 48, showing probably a reflex vagus action. Another case which has interested me demonstrates the possibility of stirring up a small lesion in the opposite lung. Twenty-four hours after injection in a cavity case, a sharp pain occurred in the opposite lung with rise of temperature. Pleuritic rubs and moist pulmonary râles appeared over an area 3 in. in diameter. These signs and symptoms entirely disappeared in forty-eight hours. This corresponds to similar cases reported by Brauer and Spengler in which they offer the explanation of a focal tuberculin reaction. It appears a possibility, but I must admit to a somewhat

anxious suspicion that it may prove to be a new and rather acute area of infection.

Dr. BALDWIN: May I make a contribution to the technique of the subject? Knowing that the depth of Drs. Floyd and Robinson's needle was sometimes a little hard to gauge, I had this (exhibited) made as a gauge for the depth of the needle. (See illustration.) Its advantages are possibly obvious in some cases, and sometimes it is not necessary. This man should have an assistant always to hold the needle. He may not need it, but in re-fillings I have been obliged to work without any assistant, and I think that in using one hand on the needle and resting this on the chest prevents the needle from going in too deep; and in the primary operation it may serve a purpose in maintaining the needle at the right depth, not allowing it to come out before the nitrogen has begun to flow in. It is also an advantage not to allow the needle to pull back.

Dr. F. M. POTTENGER (discussing papers on pneumothorax): It seems to me, as has been stated here, that this operation is one particularly for the hopeless cases of tuberculosis. One of the difficulties among those of us who practise in health resorts where we are obliged to treat large numbers of far-advanced cases, is to know when a patient is hopeless. In giving a prognosis in a patient suffering from advanced tuberculosis, I am accustomed to say that as long as the heart is able to do its work well and the patients are able to assimilate their food, there is a chance of recovery. I have fought on those principles and have won in many cases where I would have failed had I been appalled at the severity and extent of the lesion alone. In many cases I would have had a failure had I stopped at the end of six months, nine months, or even one year, but by going on for two years, or even more, I have obtained a good result. I have been observing Brauer's work in Europe ever since it began, but so far I have not done any of it. I would like to ask those who are using pneumothorax what they estimate its percentage value in arresting the disease to be, also in what per cent. of cases they have found difficulties in performing the operation?

Dr. MINOR: I have performed this operation on some twenty odd patients and feel with Dr. Otis that it is chiefly of value to us in cases otherwise hopeless and in which the chance of recovery is very small. In this type of case, in which mortality would be nearly 100 per cent., I have saved three out of twenty-one cases which were otherwise reasonably supposed to be doomed. This, in cases of this sort, is a most satisfactory result. Further, it has proved valuable to me in cases of intractable hæmorrhage. In one bad old fibroid case, subject to recurring hæmorrhages which nothing could stop permanently, I have now stopped a number of such hæmorrhages and the stoppage has lasted much longer than by other measures, although after periods varying from weeks to months, they have at times recurred, but never in as bad form as before. In my experience I

have been impressed with the danger of wakening up a slight process in the good lung by throwing on it too quickly the tax of the whole respiration and am impressed with the importance of beginning therefore with small injections and gradually increasing the amount. Further, the condition of the good lung must be considered carefully before we proceed with the operation. Naturally in cases of this sort we cannot expect it to be perfectly healthy, but roughly I would state that I would not like to see more than one-third of the other lung involved, and the process in this lung must not be very active.

The results at times, even in cases where it cannot be permanently useful, are really phenomenal. In one case with advanced trouble of a number of years' duration, with high fever, great destruction of tissue, extensive cavity formations, and all the symptoms of an advanced consumption, the injections at first had a most phenomenal effect, the temperature quickly came to normal, the pulse came to normal, the expectoration and cough almost disappeared, the appetite (which is not the rule, by the way) increased greatly and she felt like a new woman. However, this only lasted for about three weeks. At the end of this time her other lung woke up, took a rapid and spreading process, the gas had to be removed, and in two months she was dead from a rapid spread in the other lung. Referring to Dr. Griffin's cases, it seems to me that he did not control them with the X-ray. This, I believe, is extremely important, and the fluoroscope or the radiograph should be used, if possible, in all cases. It is not absolutely essential, but it will enormously handicap our efforts to be without it. Infection of the chest wall should not occur in ordinary cases, but where a pyothorax exists and the pus is withdrawn before the injection of the gas, the withdrawal of the needle will certainly infect that track. This happened to me in the case of a young girl with a pre-existing pyothorax in which pus would recollect. In drawing out my needle after tapping the pus each time there developed, one or two weeks later, hard nodules of infection at this point; it was very chronic, caused no trouble, and when she went home for her vacation she was feeling splendidly and they were not causing trouble. In another advanced case in which I tried pneumothorax, which, by the way, proved to be a failure, there has developed since the operation a fistula and there is now a pyopneumothorax which connects with the surface of the fistula and through which gas and pus comes out. Her condition has been made rather better than worse by this occurrence. Once or twice I have wounded lungs with my needle, but save for a temporary blood-spitting there has been no harm done, I am glad to say. In only one case have I done harm and shortened the patient's life, though not in a case which could have recovered. This was a case of abscess of the lung in which the pressure of the gas, since the abscess was closely under the surface, ruptured into the pleural cavity, creating an empyema which unquestionably hastened the patient's

end. On the whole, I am very well satisfied with the procedure and think it a great addition to our measures in a certain class of case, although I do not think it is going to be used except in carefully selected cases, after the first furor passes off.

Dr. ELLIOTT: Dr. Floyd has become enthusiastic over some of the good results that he has seen. Should not some of his statements be qualified; for instance, he said that some early cases recovered because of the injection. I wonder whether they would not have recovered without it. I would like to ask the question: When the fluid forms after a pneumothorax has been performed in a dry case, does it show the characteristics of an exudate or of a transudate; is it inflammatory in its character or purely hydropic? May I ask, too, where pneumothorax has resulted in rupture of the lung, the breaking through into a cavity, what pressure has been used in these cases? Has it occurred only in those cases where a high pressure has been used?

Dr. FLOYD: In regard to Dr. Otis's statement that the Germans believe that pleural shock is always due to air embolus, I would say that no gas had been injected into the patient whose case I have cited, only the needle was introduced, therefore no chance of an embolus. Nitrogen is used in preference to air because it is less readily absorbed; probably it stays in the chest two-thirds longer than ordinary air. I agree with Dr. Minor in regard to the value of the X-ray in indicating when it is time to continue with the injections, but I also believe that when you find negative pressure you have waited too long instead of not long enough. I believe 50 or 60 per cent. of unilateral cases may be arrested by nitrogen. Dr. Elliott asks why I state that early cases were arrested by this process. In the early cases that I have treated, an arrest was brought about in a very few weeks, once or twice in two weeks, that is, all the moisture disappeared. This is much more rapid loss of moisture than can be obtained by other methods. In regard to the question of effusions being inflammatory or transudates, I would say they are probably transudates.

A YEAR'S OBSERVATION OF SYMPTOMS IN CASES OF ADVANCED TUBERCULOSIS IN THE LOS ANGELES COUNTY HOSPITAL, IN CONNECTION WITH WEATHER CONDITIONS.

By CHARLES C. BROWNING, M.D.
LOS ANGELES, CALIFORNIA.

In 1908 I reported to this Society some observations made regarding the apparent influence on temperature and hæmorrhage in tuberculous patients with reference to certain meteorological phenomena.

It appeared from that report that unseasonable or very sudden changes in temperature appeared to influence temperature of patients, while equal or greater changes occurring slowly did not.

Of the cases of hæmorrhage reported at that time it appeared that hæmorrhages tend to occur in groups. Of the hæmorrhages occurring in groups about four times the number occurred when there was a barometric pressure change exceeding 0·3 of an inch within twenty-four hours than when the barometric change was less. The hæmorrhages appeared to be more frequent if there had been a decided change in one direction, as a marked rise followed by a marked change in the opposite direction—a sudden fall.

To-day I desire to call your attention to observations extending over a year on patients in the wards for advanced cases of tuberculosis in the Los Angeles County Hospital. Charts representing the scope of the work, also the different phenomena, are herewith presented. These charts, I believe, represent fairly the observations for the year.

Mr. E. J. Ellsworth collected the data and prepared the charts. The care with which this has been done is evident from the charts themselves.

He was on the ward continuously, and at my request submitted to me some of his conclusions based on observations recorded on the charts, from which I will quote later. Up to the time he completed this work he had not seen my previous report, and I purposely avoided making suggestions which might influence him.

I am also indebted to Dr. Blanche Brown for carefully reviewing the charts and submitting opinion.

The patients on this service were all in the advanced stages of pulmonary tuberculosis; most of the deaths occurring in the Hospital from tuberculosis were on this service.

There were recorded during the year twenty-one thousand nine hundred and sixty-eight (21,968) patient day observations, with an average of sixty-one patients charted daily.

There was an average of about seventy patients in the wards, but there were always a number who for various reasons were not noted.

Hæmorrhage and death groups are quite noticeable during some months, but not during others.

There were recorded two hundred and seventeen (217) hæmorrhages—six of which were fatal—and two hundred and twenty-seven (227) deaths.

Of course, climatic conditions can only act to a certain

extent as determining factors in producing symptoms recorded, and climatic conditions are always a complex of conditions, certain phenomena of which may be used as indices, these indices varying in degree of prominence with varying complexes. We may assume the following to be the average mean or normal conditions for Los Angeles:—

The barometer, which we will place at 30.

The thermometer, which we will place at 65° F.

The humidity, which we will place at 50,

and the velocity of the wind at seven miles an hour; direction, most frequently north-east land breeze at night, changing by way of north and west to south-west sea breeze during the day. Light cloudiness in the morning, clearing about nine o'clock.

Again, the subjective conditions recorded of patients are to a limited degree indices of the actual conditions of the patients, and many equally important conditions appear to observers and patients which are less easily recorded, and which do not bear a constant relation to the symptoms most prominently shown on the charts. For instance, I will quote from Mr. Ellsworth's observations. He gives two illustrative combinations:—

Combination No. 1.

"The barometer is gradually rising, the humidity slowly falling. This will nearly always give us the wind west of north, the velocity at normal or above, a clear or clearing day and more or less sunshine. The effect of this combination is remarkable. I find the patients pleasant and in some cases jovial, inclined to be optimistic as to the future; in fact, looking on their surroundings with a degree of contentment."

Combination No. 2.

"The barometer is falling either gradually or rapidly, the humidity is rising and becoming more oppressive as the hours go by; these conditions usually give us a wind from the east of north with a velocity below normal, a foggy or cloudy day with little or no sunshine. The effect on the patients of this combination is entirely different from the one first mentioned, in that they are pessimistic, cross, very irritable, some of them unreasonable in their demands. It is true that there are many other combinations in climatic conditions; sometimes they are composite of the first and second, but usually conditions are those mentioned above."

A third combination is noticeable occasionally during the winter, and results in the "Northers," as shown on December 4th and 5th.

On the 4th, in the early portion of the day, the barometer had fallen 0·20 of an inch in twenty-four hours, and began an abnormally rapid rise, reaching 0·30 of an inch during the next twenty-four hours. Clear. Relative humidity fell from 75 to 20 within twenty-four hours and to 8 during the following day. Temperature, practically unchanged. Wind, north-east, north and north-west; mean velocity on the 4th, ten miles; on the 5th, nine miles. This condition is accompanied by a marked drying of the mucous membrane, causing great discomfort in some and comfort in others. There is a generally increased nervous tension, which is exceedingly disagreeable to those most susceptible, and an agreeable amount of stimulation in the phlegmatic. These nervous phenomena are quite noticeable even in the lower animals.

I regard the temperature records in this report of less value than in my former report. In that report the temperatures reported were taken by the patients and could be taken at regular intervals. In this series they are taken by

the nurses, which renders it impracticable for them to be taken as regularly.

The conditions which appear to influence groups of hæmorrhages and deaths appear to be barometric pressure, humidity and cloudiness—each in its turn appearing to be the most prominent index to weather conditions, which seemed to exert a limited determining influence, as shown by the charts herewith presented.

I have chosen the charts for eight months, and believe they are typical of conditions. The ninth chart shows the tuberculosis death-rate of Los Angeles as a whole, by months together, with certain weather conditions.

The photographs do not show the same shading throughout for clear, fog and cloudiness, for the reason that in keeping the charts different colours had been used to represent these, and the same colours were not used continuously to represent the same conditions. In some of the months colours which photograph black were used to represent more than one condition.

Fog.

The term fog is generally used to convey the idea of a cold or chilly condition of excessively moisture-laden atmosphere. This condition is occasionally met with here, but the prevailing condition noted as fog is the one generally spoken of locally as "high fog." No effort has been made to differentiate between these. The "high fog" is regarded by many as one of the most desirable factors of the Southern California climatic condition.

Ford A. Carpenter, of the United States Weather Bureau, a corresponding member of this Association, in "The Climate and Weather of San Diego, California," states, in referring to this condition :—

"It is not fog in the generally accepted meaning, for

this 'light veil' is neither cold nor excessively moisture-laden. Neither is it high, for its altitude is less than a thousand feet."

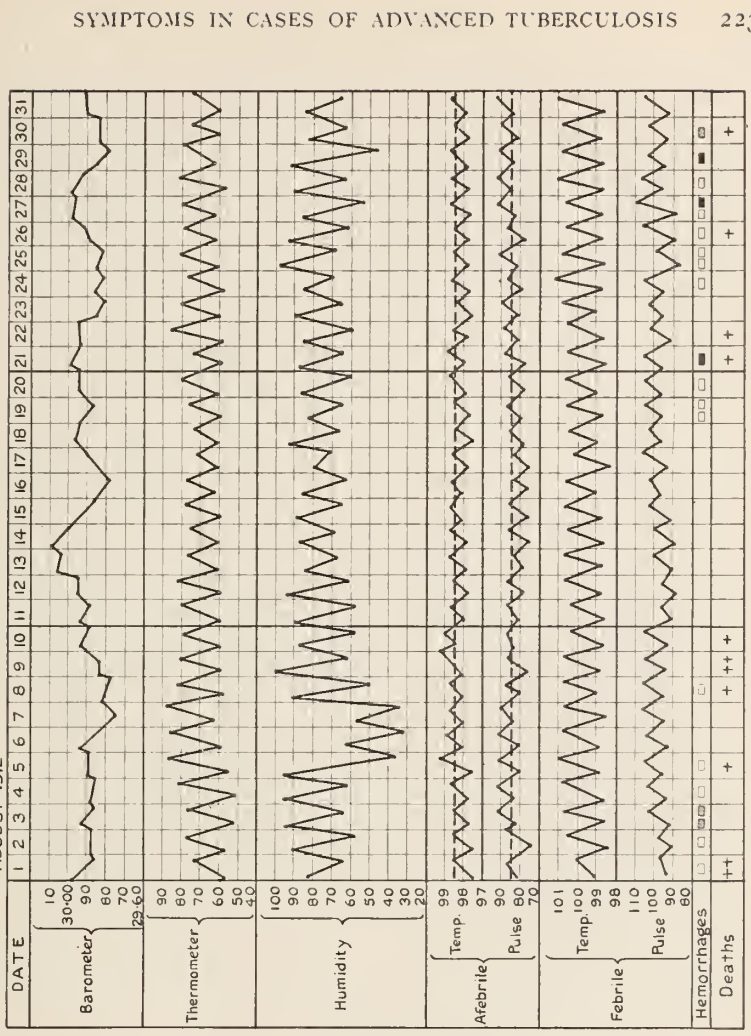
"To one who has spent a few weeks of spring, summer, or fall in Southern California, the picturesque description of the musical Spanish *el velo* is quickly recognized as both expressive and truthful."

The velo cloud is expressive of the diaphanous cloud which, of a midsummer's morning, shades the brilliant sun until such time as the veil is swept aside by the ocean breeze, whose cooling breath renders it no longer necessary for a delightful day. This condition is beautifully expressed by some quotations by Mr. Carpenter:—

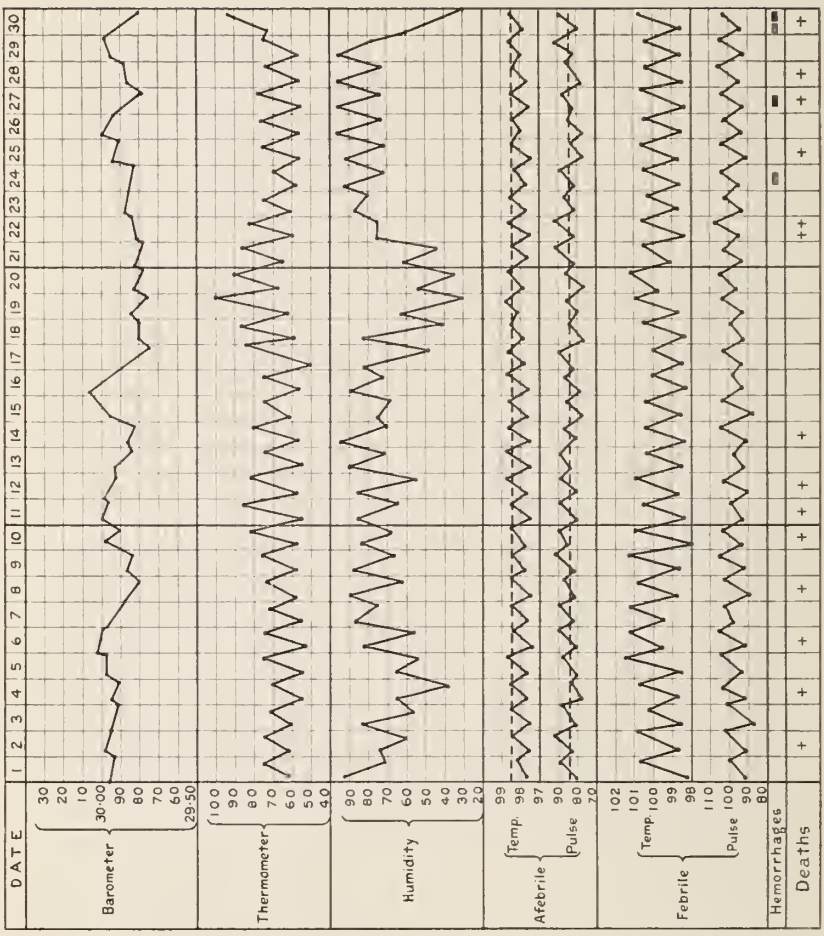
"*El velo de la luz*": "The veil that hides the light." "*Velo qui cubre la luz del sol*": "The veil which shades (covers) the light of the sun." "*El velo de la mañana*": "The veil of the morning."

"The screening of this region from the sun's rays is so thoroughly accomplished that during a normal summer's day (April to November) the sun breaks through the velo cloud about ten o'clock, the sky clearing shortly afterwards and remaining free from clouds until about sunset."

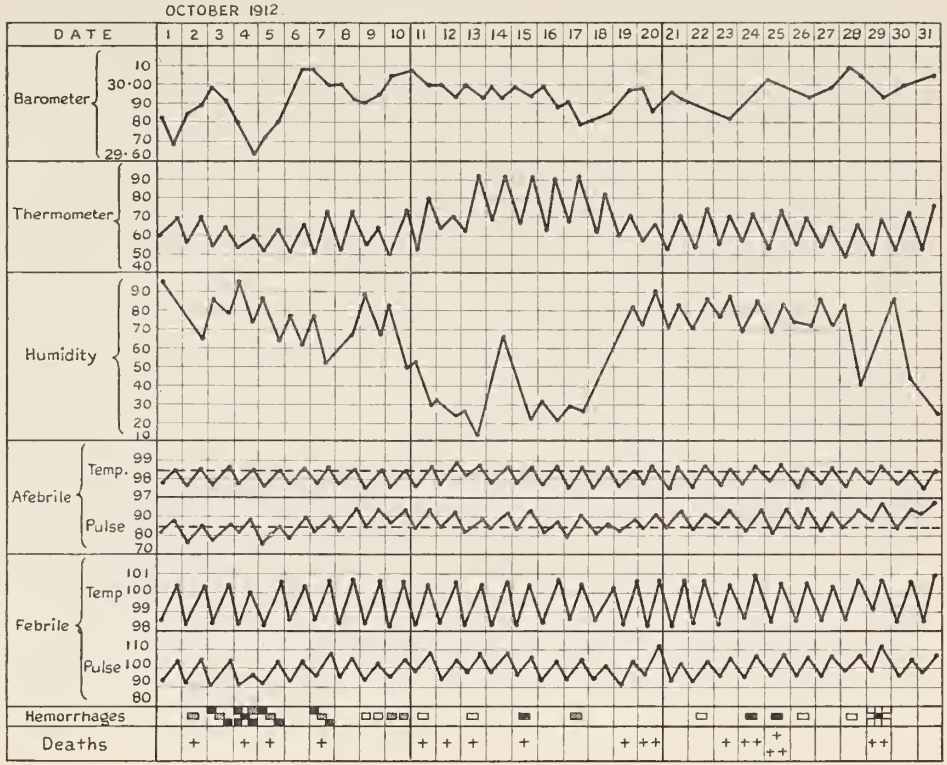
I beg to present the following charts, which I believe to be self-explanatory:—



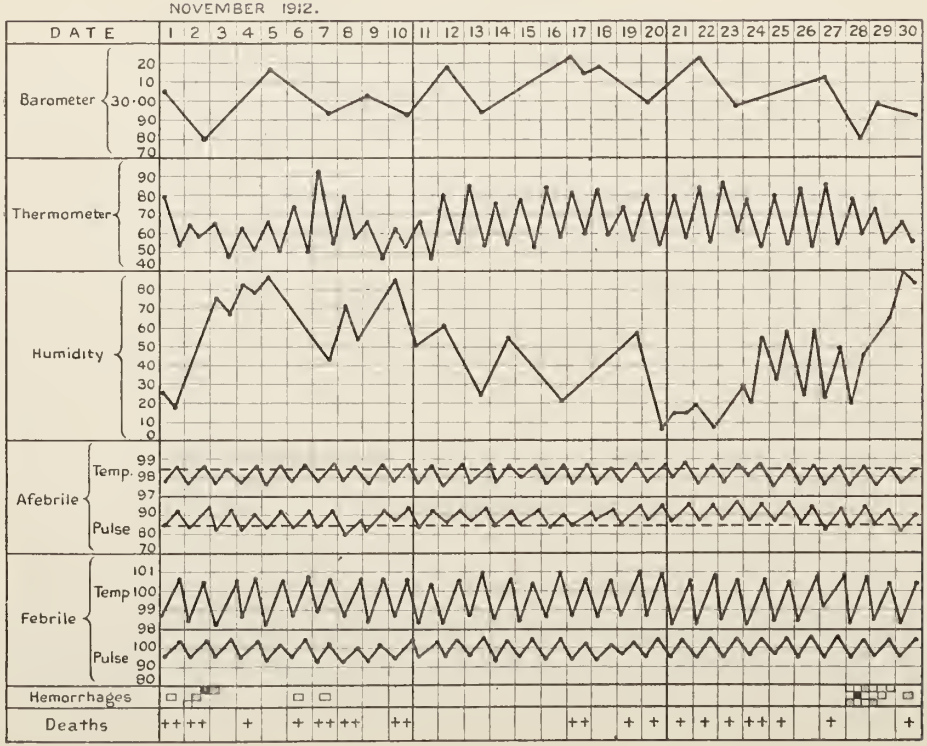
Daily average number of patients, 61. Ordinary summer month—freedom from severe hæmorrhages or grouping of deaths.



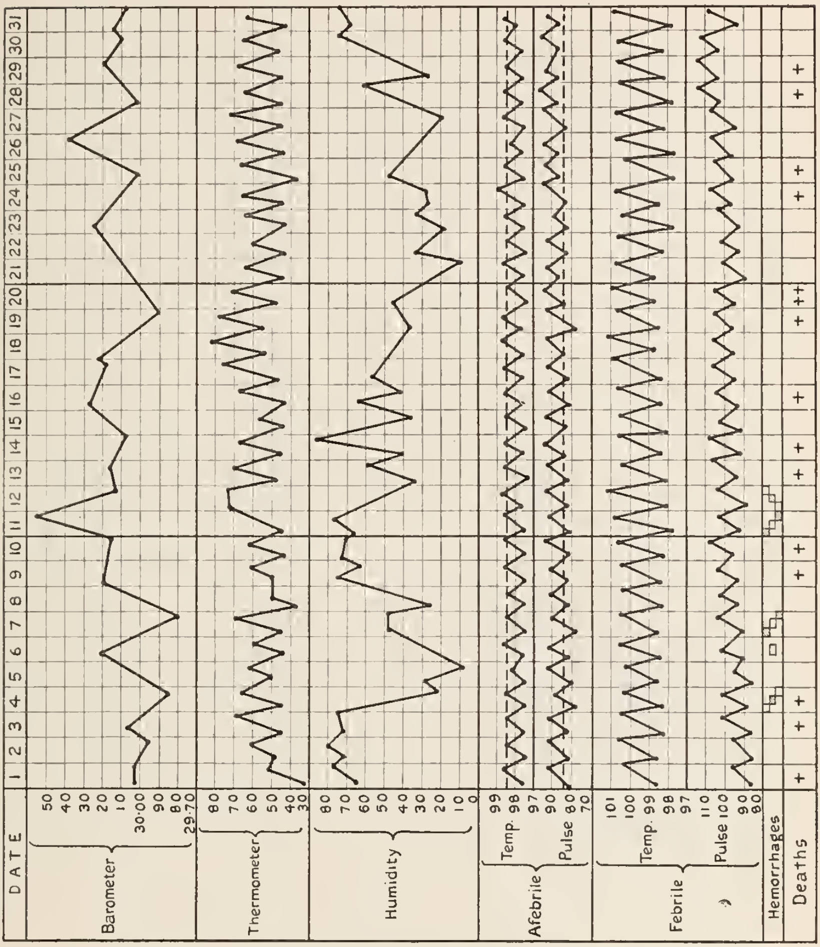
Daily average number of patients, 62. This chart shows no marked meteorological changes. Note evenness of distribution of deaths, few hemorrhages and freedom from groups.



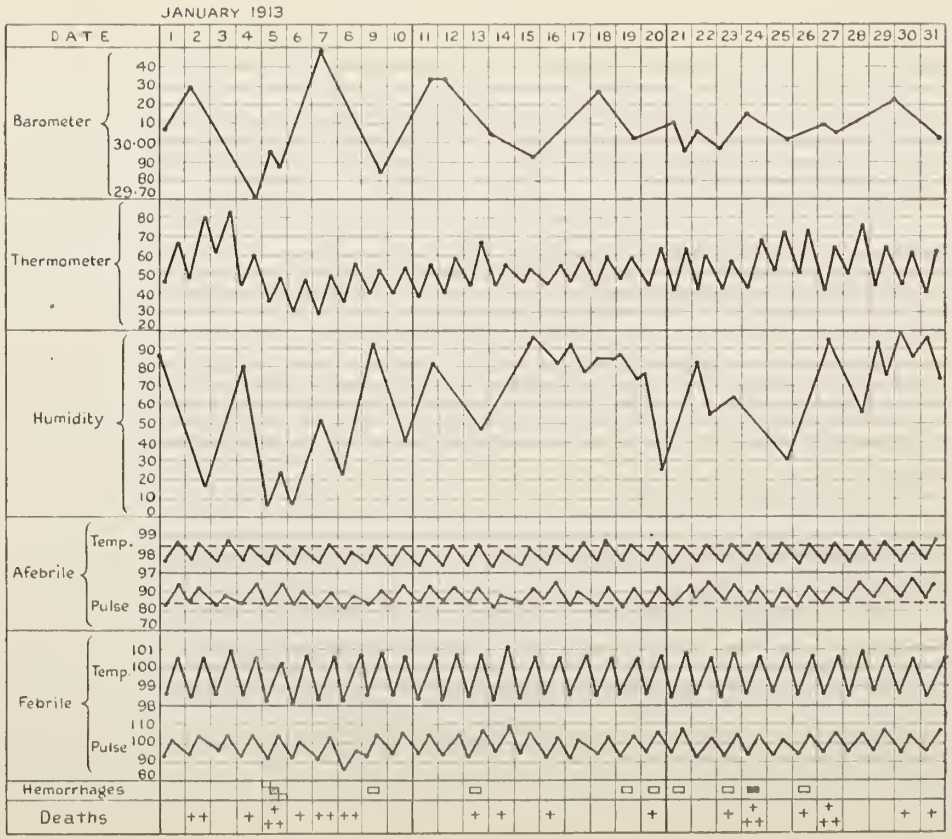
Daily average number of patients, 66. Early part shows group — especially hæmorrhage — accompanied by extreme reverse barometric changes. The latter portion of month shows group of deaths and few hæmorrhages accompanying moderate barometric change. Humidity relatively high. Cloudy.



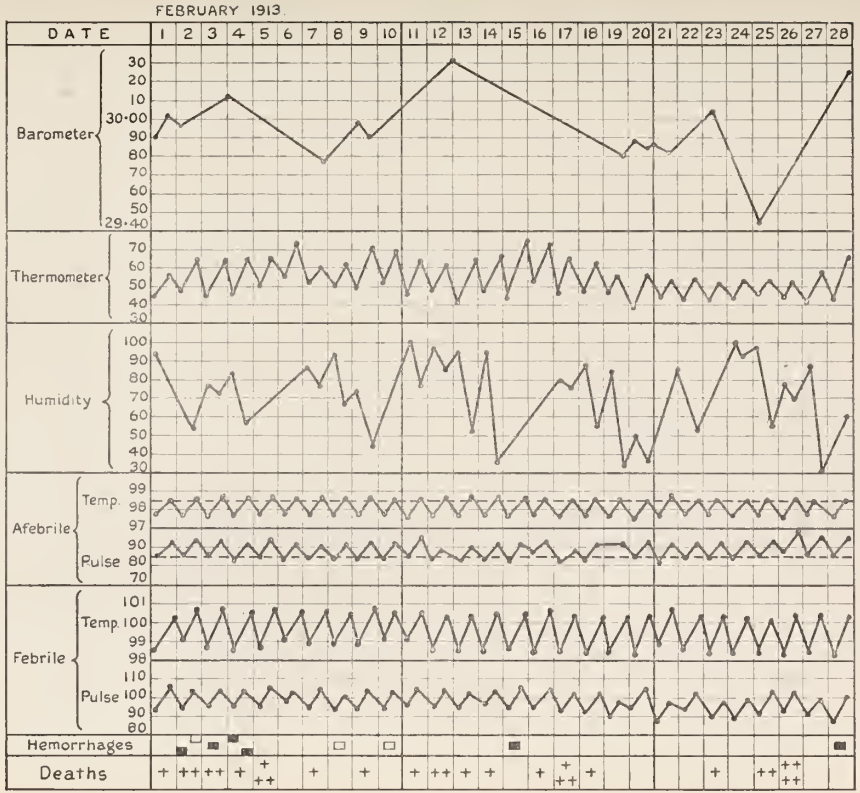
Daily average number of patients, 65·4. Early and latter portion of month show sudden barometric changes, humidity high, temperature moderately low, with group hæmorrhages and deaths. The middle of the month barometric changes equal to former, but taking place slowly. Temperature higher, humidity lower, free from hæmorrhages, deaths occurring regularly.



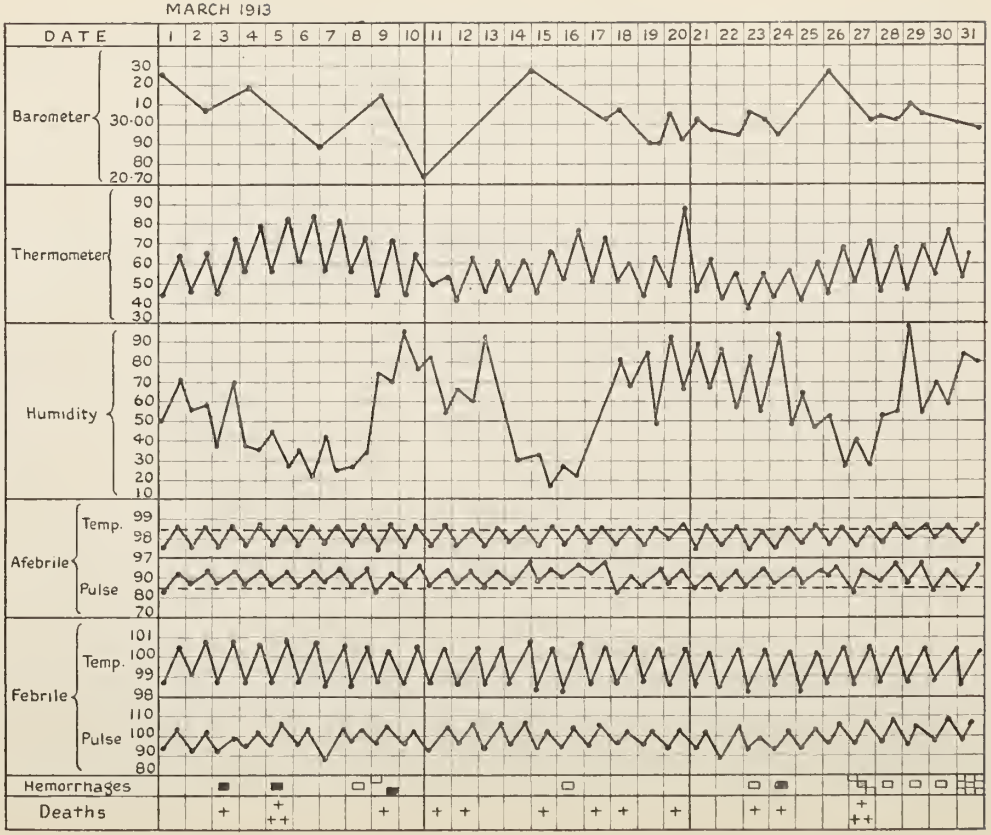
Daily average number of patients, 68·4. Early portion of month extreme sudden changes. Later in month extreme | gradual changes.



Daily average number of patients, 85. Early portion of month extreme changes followed by moderate changes. Ordered patients with mild pleuritic pains kept quiet and chests bandaged or strapped. Had observed that frequently patients complained of slight discomfort preceding hæmorrhage.



Daily average number of patients, 86·6. A winter month free from groups of hæmorrhages, excepting early. These were all in one patient who refused treatment. He died on the 15th, following a profuse hæmorrhage on the 14th inst.



Daily average number of patients, 71. Early part of month suggests barometric changes as index, while latter part high humidity and low temperature are suggested. Four fatal hemorrhages during this month without marked weather changes.

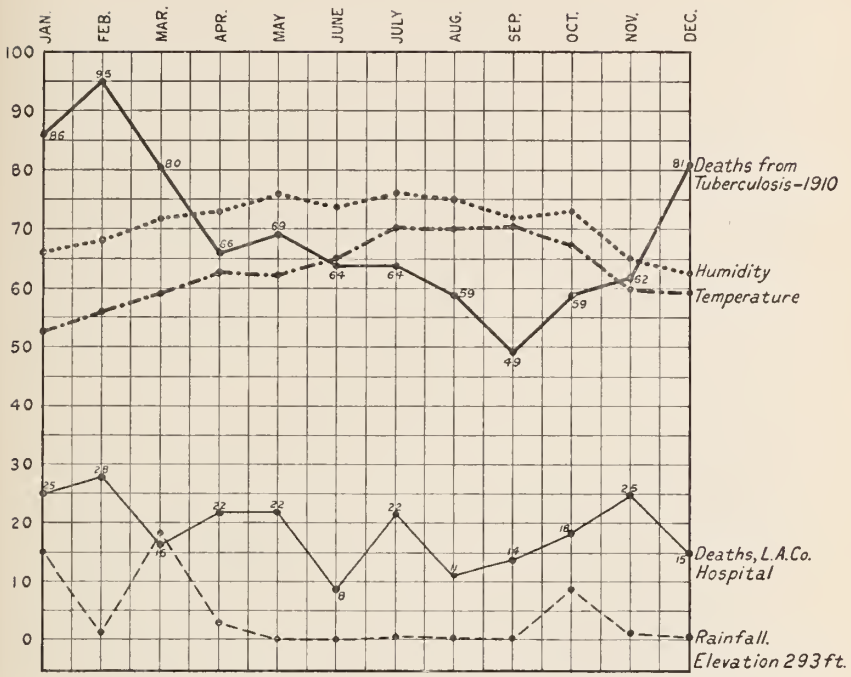


Chart Showing Relation of Temperature.

DISCUSSION.

Dr. F. M. Pottenger: Since these studies have been published by Dr. Browning, I have often wondered if similar observations made at other places would show the same relationship. I have noticed that everything goes by groups in a sanatorium. In the institution with which I am connected we have about eighty or ninety patients. I can usually tell in the morning, after seeing the first few patients, what the general condition of the patients will be for that day. Sometimes we see the entire number without any unusual complaints. Other times probably fifteen to twenty-five will have the same disturbance, either pleurisy or pains in the chest, expectoration of blood, rise of temperature, &c. I have sometimes wondered whether it is more difficult to reduce the temperature in a warm country than in a cold country. If we have a sudden rise in daily temperature, say, the maximum of the previous day was 60° F. and it rises to 75° or 80° F., the patients on the first day suffer more from the heat than they do on the following days, even though they be hotter. It seems that heat dissipation adjusts itself to a certain point. If we have a sudden

rise the heat-regulating mechanism cannot adjust itself quickly, therefore the body temperature will rise more than it will if it has had time to adjust itself. We notice a difference of from one-third to one-half or even a whole degree on the first warm day following a cold spell. The succeeding days, however, may be even warmer and yet the temperature does not go any higher or so high as the first day. If there is a sudden drop in temperature we note the reverse. With the various changes in the weather conditions and barometric pressure, we always note that pleurisy, neuritis and hæmorrhage are more apt to occur. If one of my patients is having a severe hæmorrhage and I see a fog coming in I feel that the patient will probably have another hæmorrhage. If an electric storm comes up I also feel that the patient will probably have further hæmorrhages. If one watches carefully he can almost predict the way hæmorrhage patients are going to go by the weather conditions.

Dr. BROWNING: I have nothing further to add, except to say that it is the sudden extremes in temperature, in barometer pressure and humidity, that are serious. It may be surprising to some of you to know that twice last year the relative humidity in Los Angeles was as low as 8 per cent., and it not infrequently goes as low as 20 per cent., and we have that high fog which is popularly known there and referred to in Mr. Ford Carpenter's book and quoted by Dr. Carrington as the "velo," the veil cloud, which influences the atmosphere materially, and to most people tempers the climate greatly. There is an atmospheric condition, frequently spoken of as the "Norther," which is exceedingly stimulating to some, even to the point of irritation, and to the phlegmatic it brings a stimulus to their best.

Dr. DARLINGTON: I have found that by forcing the drinking of water, as we do in the steam mills, by giving a great deal of water to a man with a chronic grouch, we can relieve it.

THE INFLUENCE OF SMOKE ON ACUTE AND CHRONIC LUNG INFECTIONS.

By WILLIAM CHARLES WHITE and PAUL SHUEY.
PITTSBURG, PENN.

A year ago we started an investigation on the influence of smoke on the various problems of public health. We chose for our study the respiratory tract, as that in which smoke would have its greatest influence by direct contact.

The investigation was carried on under a grant given by Mr. R. B. Mellon to the University of Pittsburgh for the consideration of this problem from all its aspects.

At the International Congress of Hygiene and Demography we made a preliminary report of what had been accomplished up to that time, and while the work reported in the former paper was very incomplete, there seemed to be a rough direct ratio between the number of smoky days in any given city and the number of deaths occurring from pneumonia. On the other hand, there seemed to be an inverse ratio between the number of smoky days and the number of deaths from tuberculosis.

In choosing these two diseases, we were guided by the fact that the one (pneumonia) is an acute inflammatory process of short duration, likely to be influenced by acute irritation, such as would come from foreign particles, and that it represents the most striking malady from the stand-

point of increased mortality in the greater number of our cities.

The other (tuberculosis) is a chronic infection, characterized by infiltration and healing by fibrosis, extending over a long period of months or years, and more likely to be stimulated to healing by irritation of inert foreign particles, and yet presenting a constantly decreasing mortality.

Following the presentation of the paper before the Congress of Hygiene and Demography, Dr. John S. Fulton, Secretary of the Congress, called our attention by letter to certain factors, which he felt were of more importance than the simple ratio of smoke to pneumonia mortality, and we have included these in the substance of our present study. The main points in Dr. Fulton's arguments against the conclusions which we tentatively drew from our study were as follow:—

"I would expect Boston to have a greater mortality from pneumonia than Pittsburg, on the sole basis of fact that Boston has relatively more people in the pneumonia ages. I would expect Chicago to have a higher pneumonia mortality than Pittsburg, because there is a pneumonia obsession in the minds of the medical profession of Chicago.

"Chicago has relatively more people in the pneumonia ages than Pittsburg has, and relatively fewer in the pneumonia ages than Boston. I do not think that the pneumonia figures admit of sound reasoning as to magnitude, unless, in the first place, distinctions are made as to the age-distribution of the populations which are to be compared; and, in the second place, unless the pneumonia mortality is divided sharply into two groups, those under and those above the age of three years, and the comparisons made with reference to these distinctions.

"A comparison of pneumonia and tuberculosis magnitudes, as among the cities which you mention, does not

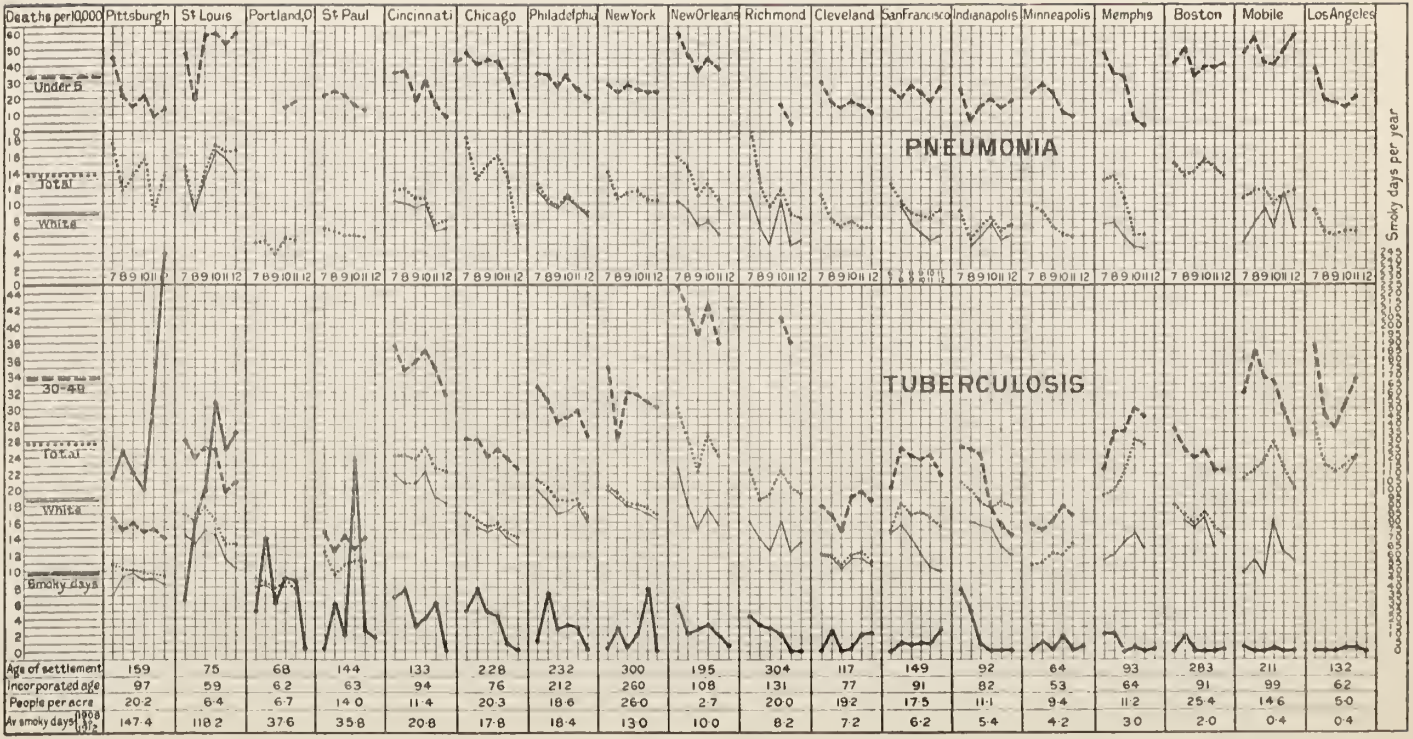
prove that the prevalence of tuberculosis in Pittsburg is low, or that the prevalence of pneumonia is high.

" By mere inspection of those pneumonia charts, without any key to the names of the cities concerned, I would say that 1, 2, 3, 4, 5, 6 and 7 (Chicago, Pittsburg, Boston, New Orleans, Richmond, New York and Charleston) are cities fifty years old or older. The last eight (Cincinnati, St. Louis, Philadelphia, San Francisco, Mobile, Cleveland, St. Paul and Portland) are cities less than fifty years of age, and probably situated west of the Alleghenies. I would be right with respect to the first group, and, with respect to the last group, my two errors would be Philadelphia and Mobile."

In choosing the cities for study, we have taken the larger cities scattered widely over the United States, and have tried to get as widely varying conditions from the standpoint of age of settlement, density of population, years of incorporation, flatness of contour as it was possible to obtain.

We have also analysed more carefully the data on which we have completed our present study, and have ruled out as much as possible, in drawing our conclusions, the years and material which were unsafe to use by virtue of their lack of thoroughness.

In the charts accompanying the present study, in the smoke curve we have plotted the number of smoky days per year. In arranging the cities on the charts we have put the smokiest cities first, and so graded on down to the cities in which the smoke was the least. We have considered in arranging them in this way only the 1908 to 1912 periods, preferring to neglect 1907 on account of the unreliability of the mortality data of that year. It will thus be seen that Pittsburg, St. Louis, Portland, St. Paul, Cincinnati, Chicago, Philadelphia, New Orleans and New York may be grouped as the smoky cities. In this group, however, Pittsburg, St. Louis, Portland and St. Paul may be classed as very smoky cities.


Deaths per 10,000
Pittsburgh
St Louis
Portland,O
St Paul
Cincinnati
Chicago
Philadelphia
New York
New Orleans
Richmond
Cleveland
San Francisco
Indianapolis
Minneapolis
Memphis
Boston
Mobile
Los Angeles
Under 5
Total
White
PNEUMONIA
30-49
Total
White
Smoky days
TUBERCULOSIS
7 8 9 10 11 12
Smoky days per year
Age of settlement 159 75 68 144 133 228 232 300 195 304 117 149 92 64 93 283 211 132
Incorporated age 97 59 6·2 63 94 76 212 260 108 131 77 91 82 53 64 91 99 62
People per acre 20·2 6·4 6·7 14·0 11·4 20·3 18·6 26·0 2·7 20·0 19·2 17·5 11·1 9·4 11·2 25·4 14·6 5·0
Av smoky days 1908 to 1912 147·4 118·2 37·6 35·8 20·8 17·8 18·4 13·0 10·0 8·2 7·2 6·2 5·4 4·2 3·0 2·0 0·4 0·4

Beneath these charts the figures represent :—

First (the top line), the number of years since the settlement of the city;

The second line, the number of years since the city was incorporated;

The third line, the density of population on the basis of the number of people per acre; and the

Fourth line, the average number of smoky days per year for the five-year period, 1908-1912.

In studying the death ratio of the different cities, we plotted the total death-rate and the death-rate of the white population, as well as the following groups :—

Total population under 5 years.
,, ,, from 5 to 9 years.
,, ,, ,, 10 ,, 19 ,,
,, ,, ,, 20 ,, 29 ,,
,, ,, ,, 30 ,, 49 ,,
,, ,, ,, 50 ,, 69 ,,
,, ,, ,, 70 years upwards.

While we plotted all these in the original chart from which this study was made, we have only included a few of them outside the total death-rate in the accompanying charts. In these curves, it is important to bear in mind the history of the registration area of the United States and the great variation in the thoroughness with which vital statistics are kept in the various cities. It was only in 1907 that the registration area approximated 50 per cent. of the population; and only in 1910 was any special attempt made at a uniform method of classification and registration, and this uniform method is not yet in operation in all of the municipalities which we have used. For instance, this classification is not in use in Philadelphia, nor in St. Louis.

In the various curves it will be observed that the year

1907 does not conform to the ratio which has been found to hold in years following this. This may safely be explained by the fact that 1908 was really the beginning of better attempts to place vital statistics upon a more uniform basis; and only in 1910-1911-1912 did we arrive at any real fair basis for comparison.

It must be borne in mind that in comparing with other cities, cities having a large coloured component of population, the mortality by age must be compared on a basis of the ratio of the mortality of the white population to the entire population, because the negro death-rates are much higher than the white.

It will be obvious immediately to anyone who attempts to study this field that the number of smoky days, as furnished by the United States Weather Bureau, is apt to be a very variable quantity, both from the personnel of the recorder and the method by which these readings are made.

The present method of determining the number of smoky days is by fixing the clearness with which certain established objects can be observed from the fixed point of the Observation Bureau. Such factors as the height of the Bureau from the ground, the acuteness of vision of the observer, probably the habits of the observer, the interest which the observer has in the problem, and similar circumstances make it almost impossible to lay down any fixed curve which will be comprehensive for all cities, and it would seem a reasonable conclusion that if there is even a rough relation between the conditions which we are studying, it would be safer to say that with more careful figures a more intimate relation could be determined. For instance, it is inconceivable that there should be no smoky days for 1910-1911-1912 in a city like Boston, which is largely a manufacturing centre. In the report of the District Forecaster for Boston, there have only been two smoky periods in five years. These two periods

were five days following September 1, 1908, and five days following October 15 in the same year, and were due to forest fires. While it seems almost incredible that in a manufacturing city, subject to fogs, there should not be more smoky days than is indicated by the Forecaster's report, yet it will be noticed that in the Boston curve during this smoky period the pneumonia death-rate decreased It is a feature probably not of very serious import that during that year the pneumonia death-rate fell in all pneumonia groups save that of 5 years of age. This evidence of the reverse influence of the number of smoky days per year on the mortality curve from pneumonia is apparent in several other cities, as for example, Cincinnati.

Between St. Paul and Minneapolis, lying very near together, there is also a marked discrepancy—St. Paul having in 1910 nearly 120 smoky days, and yet the reply from the Forecaster was as follows: "You are informed that said days with record of smoke had reference only to smoke in the atmosphere due to distant forest fires, &c., and not to local smoke due to factory chimneys, &c." It seems impossible that St. Paul should be affected with forest fires and Minneapolis not, with a few miles' difference in position. This peculiarity of the smoke curve for St. Paul would, of course, put it out of place in this chart, and would explain the difficulty in tracing the same relation which is present in the other cities.

The same thing applies to Portland, from which we have not been able to receive an explanation as to the reason for the large number of smoky days reported from that city; but Portland, also, is probably out of its place in this scheme, and the statistics are not complete for this city.

In summing up these charts, which have been done quite impartially, the only constant factor which seems to have any relation is the smoke; in other words, where age of

settlement, number of people per acre, and age of incorporation have any apparent influence, this influence must be coupled with the number of smoky days before any satisfactory conclusion can be drawn. It will be seen, then, that if we except Portland and St. Paul, there is a general tendency of the tuberculosis death-rate to rise as the number of smoky days in the city decreases. On the other hand, it will be seen that there is a general tendency for the number of deaths from pneumonia to fall as the number of smoky days in the city decreases. In this instance, also, Portland, St. Paul and Boston must be excepted. There seems to be no definite relation, however, between the number of smoky days and the death-rate under 5 years of age in the pneumonia group. This might readily be expected if we consider as the explanation of the influence of smoke on pneumonia the irritative changes which go on in the mucous membrane of the upper air passages as the underlying factor in this relation, and that these changes would probably take years in their production, or, as Dr. Haythorn has shown, the pneumonia difficulty may be largely one of absorption of exudate, which anthracosis by plugging the lymph spaces largely impedes.

In general, the tuberculosis age-groups are rather uniform in their relation to each other when one comes to the study of individual influences; probably nothing is more striking than the difference between the curve for the total death-rate of the white population as opposed to the coloured. This is most strikingly seen in such southern cities as Memphis, Mobile, New Orleans, and Richmond. There is a striking difference, also, in San Francisco and Los Angeles in the total death-rate from tuberculosis, due, likely, to importation from the middle-west and northern parts of the country.

When one studies individual cities, one finds, as in

Pittsburg, St. Louis, Cincinnati, Chicago, New Orleans, Richmond, &c., a noticeable similarity between the total pneumonia death-rate and the total number of smoky days. This is almost entirely absent in comparing the tuberculosis yearly death-rate, which has persistently dropped in most of the individual cities, save the southern ones, in which there have been curious rises. It is not our intention to enter into explanation of this feature in this paper.

We are at a loss to explain the high mortality rate from tuberculosis in Cincinnati, which seems to be out of its place in the general contour of this chart.

In Boston, in addition to the fact that we believe it out of place from the number of smoky days from a manufacturing standpoint, Dr. Fulton had suggested in his criticism of our former paper that the high pneumonia death-rate in Boston was probably due to the large number of people in the pneumonia ages (extremes of life). This our age grouping has not demonstrated, as the pneumonia death-rate in all ages is high in Boston. We believe that the factor which is absent in the compilation of this city is the number of smoky days in the year.

Chicago, on the other hand, where Dr. Fulton believes there is a pneumonia obsession in the minds of the physicians, follows very closely what one would expect from the readings of the smoky days. As nearly as we can find, Chicago has been very careful, and since 1910 has forwarded its certificates to Washington, where they have been classified by the Vital Statistics Division of the Census Bureau in order to obviate the reflection of local bias.

We believe that if it were possible to establish a reading of smoky days on the basis which Dr. Benner has established in Pittsburg, *i.e.*, the precipitation of soot, and have this uniform in the various cities, that we would be able to establish a much more intimate relation between the number

of smoky days and the number of pneumonia deaths in any city.

One of the conspicuous things to us in Pittsburg has been the virulency of the pneumonia infection, which, of course, varies from year to year, but seems to carry its toll off more quickly in Pittsburg than in any other of the four cities in which I have lived and worked in this field.

It may be well here to again call your attention to the fact that pneumonia is in the main an increasing death-rate in many cities and in the country as a whole; that it takes its victims from the extremes of life; and also takes off many of our most useful middle-aged business men, *i.e.*, many on whom most has been spent in education, at a time when they are most useful to the community; and if it were possible by municipal ordinance to control in some way the production of useless smoke in the cities, much might be done to conserve that on which the community has expended the most, and from which it may reasonably expect returns in place of death by a rapid illness, such as the one with which we are dealing.

Source of Data.

The mortality statistics are based on reports received through the courtesy of Dr. C. L. Wilbur, Chief of the Division of Vital Statistics of the United States Census Bureau, and also through the courtesy of the various Boards of Health of the different cities.

The population statistics and age-distribution for 1910 were obtained through the courtesy of Director E. Dana Durand, of the United States Census Bureau.

The smoke data we obtained through the courtesy of the Chief of the United States Weather Bureau.

Since the Census Bureau report for the year 1900 was as of the population on June 1, while the Census Bureau report

for 1910 was as of population on April 15, we utilized the method used by the Census Bureau in estimating the population figures for intercensual years, and after determining the rate of increase, we reduced the estimates of populations to a uniform mid-year basis, *i.e.*, we have them to relate to July 1, with the exception of San Francisco, in which our mortality figures were for the fiscal year. For this city we took the population as of January 1.*

It was necessary to plot population curves to provide a comparison between the population statistics which are furnished on the basis of ten-year periods, starting with five as its unit digit after 35. The mortality statistics, on the other hand, are furnished on the basis of ten-year periods with zero as the unit digit above 30 years of age.

After the population statistics were plotted on this basis, computation was made from these curves for the age periods corresponding to the mortality statistics. The mortality rate per 10,000 was then computed and used in the building up of the curves of the other charts.

DISCUSSION.

Dr. F. M. POTTENGER (Los Angeles, California): Regarding carbon and tuberculosis, I would like to mention the fact that several years ago when travelling in Europe, there happened to be a layman in the same compartment with me. As we were travelling through the coal regions of Westphalia, this man made the statement that the coal miners in Westphalia were particularly free from tuberculosis, and asked me to give him an explanation, which, of course, I could not do. One criticism of Dr. White's paper. It seems impossible to draw any conclusions whatever from isolated data of this kind. Take for example Los Angeles. According to his chart, Los Angeles has a very high death-rate, but very little smoke. The reason there is little smoke is because the factories are few. The reason the tuberculosis death-rate is high is because so many patients come into Los Angeles

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* *Bulletin* 108, p. 9, and *Bulletin* 109, p. 9, United States Bureau of Census Department of Commerce.

to die. Over half the patients who die of tuberculosis in Los Angeles die with a residence of less than a year, and, in fact, a very large percentage of them die within the first two or three months of their stay, being practically dead before they come. It would be erroneous to draw the conclusion that her high death-rate from tuberculosis is because of the lack of carbon in the air.

Dr. CASSELBERRY: So many things contribute to the causation of tuberculosis—the density of population, housing, and working conditions, sunshine, winds, and dust, aside from the carbon of smoke, that it does not seem likely that a chart, based alone on carbon contamination, can be reliable. Also it is a question whether the unhealthful effects, if any, can be ascribed directly to the carbon itself, rather than to the associated gases, carburetted hydrogen, sulphuretted hydrogen, &c., and other vitiating substances of a smoky atmosphere.

Dr. BROWNING: It would seem to me that in the open tuberculosis resorts some effort should be made to segregate the persons who die in these resorts into those who have recently become residents and those who have been longer resident. I think this would not be very difficult with reference to Los Angeles. What Dr. Pottenger has said may seem as a joke, but it is a fact. Not only what he said about half of the patients dying within the first year of residence, but about one-fourth during the first month; so you see that the climatic condition of Los Angeles with reference to any local condition has no effect upon that class of cases. I take the same to be true of any other open resort. It would be very little effort, I think, for the State Board of Health or the city health authorities to segregate them. In fact, I think this is done in many instances. I quite appreciate the effort Dr. White has made, and I know that he will feel sure that we would welcome any effort to eliminate what might appear to be factors of disease.

Dr. LINSLY WILLIAMS: Just one point on the question of pneumonia statistics, which is whether the mortality in Pittsburg was for one year period, or whether over a number of years. We find in New York the mortality in one year is higher than other years. Dr. Pottenger also speaks of the death-rate from tuberculosis in the different cities; it always seemed to me quite necessary that the cities, if possible, should have a corrected death-rate, but I do not see quite how they can figure it out if cases are reported as tuberculous and then are lost track of by removal; and it is difficult to trace them. It is rather easier in places like Los Angeles, perhaps, and other large cities where they have a good deal of tuberculosis coming, easier for them to tell where they come from than it is to tell where they went to. It may be that one of the reasons why the death-rate is diminished in the cities is because these patients are leaving the cities.

Dr. J. M. ANDERS (Philadelphia): To my mind this is a very serious subject, both from a practical and scientific standpoint. I

was much surprised to learn that the influence of smoke on the incidence of pneumonia was greater than on that of tuberculosis. It is quite possible that Dr. White's explanation of this discrepancy is entirely correct. I think it is almost an invariable rule that a person suffering from advanced anthracosis, coming from the coal regions of Pennsylvania to our hospitals in Philadelphia, finally develops pulmonary tuberculosis, which is apt to pursue the usual course of this disease. It is possible that the implantation and the progress of the disease may be retarded in the manner explained by Dr. White. Dr. White chose wisely when he selected pneumonia and pulmonary tuberculosis as types of diseases for comparison, and I think we must allow that his tentative conclusions have more than ordinary value in view of the fact that he has taken into account a great many interfering conditions, for example, the topography, the local congestion of the population, &c. He said nothing about dust in his remarks. Not wishing to criticize his paper, I still think that this and other associated impurities, as Dr. Casselberry has said, might have been considered in this connection. Truly Pittsburg, owing to its peculiar topography and the fact that it is *the* smoky city of our country, is a very fertile and appropriate field for investigating this subject, and I certainly trust that Dr. White will continue his work in this line because it will require observation over a long period of years to give us reliable results.

Dr. HINSDALE: I have always understood, Mr. President, that one of the deleterious influences of smoke was the fact that it contained so much sulphuric acid; and there are many interesting calculations about the number of tons of sulphuric acid which are deposited in a large city like London each day from the various furnaces and fireplaces in that city; so that the carbon particles being associated with free sulphuric acid, and being inhaled into the lungs, is a very bad combination, of course. It would take a long time to convince me that carbon particles even free from sulphuric acid, if such were the case, would not do the lungs any harm, or would not incite probably the infection of tuberculosis.

CLINICAL OBSERVATIONS ON BLOOD-PRESSURE.

By JUDSON DALAND, M.D.
PHILADELPHIA.

The frequency of errors in the making and interpretation of blood-pressure records suggested the desirability of bringing this subject before our Association for discussion.

The small spring sphygmomanometer is employed by most physicians, but the mercury manometer should be preferred because of its greater accuracy. Diastolic pressure observations are often omitted whereby valuable information obtained from a study of the low and mean pressure is lost. Usually, but a single blood-pressure record is made, although it is often necessary to observe the effects of work, rest, sleep and emotional excitement.

Blood-pressure depends upon: (1) The amount of resistance offered by the capillaries and arterioles; (2) the elasticity of the arteries; (3) the force of the heart; (4) the amount of blood in the arterial system, and but the slight importance is to be attached to the viscosity of the blood. The most important factor, clinically, is the state of the vasomotor system. This system may be considered from the standpoint of the vasomotor centre in the medulla, local vasomotor centres controlling regions of the body, as, for example, the brain or the gastro-intestinal tract, and the

nervous mechanism controlling individual or groups of capillaries or arterioles. These centric and peripheral centres are readily acted upon by stimuli from various parts of the body, as, for example, the cerebral cortex, the skin regions, the seat of intense pain, and also by toxæmias, variations in the secretions of the thyroid, the adrenals or the ovary.

Physiologically, blood-pressure varies at different times of the day, and is influenced by rest, cerebral or muscular work, and emotional or psychical excitement. Variations thus induced are modified by disease. A sudden unexpected noise, such as produced by a china bowl falling to the floor and breaking with a loud crash, has produced a sudden temporary increase of blood-pressure of from 20 to 30 mm. Furthermore, it is evident that a correct interpretation of blood-pressure records, more especially in those of a nervous temperament, requires not only the selection of a proper instrument and correct technique, but also that the patient be under average ordinary physiologic conditions. Multiple blood-pressure examinations, under various conditions, are often necessary before sufficient data is secured upon which to base a proper interpretation. Idiosyncrasies regarding blood-pressure may be expected analogous to those observed in the pulse-rate, the respirations and the temperature.

A man engaged in heavy manual labour, when confined to bed for some weeks from a non-febrile disease, not infrequently shows secondary hypotension, due to prolonged rest, muscular inactivity and relaxation; whereas such patients, after a day's work, show a higher blood-pressure, which would be still higher while at work.

Occupations involving great responsibilities and strain of the nervous system tend to hypertension, and the blood-pressure of an orator of a nervous temperament is apt to be high while delivering an oration. Many surgeons show

hypertension owing to the great responsibilities and excitement attending serious operations.

Temperament plays an important *rôle* in the production of variable and high blood-pressure, the phlegmatic showing the least and the neurotic the maximum variations. Extraordinary variations, all tending to hypertension, are observed in the neuropathic. Driving an automobile for two hours through crowded streets caused an increase of 30 mm. in the systolic blood-pressure and 15 mm. in the diastolic; whereas under similar conditions, while driving quietly where there was but little traffic, caused an increase of but 10 mm. in the systolic and a decrease of 5 mm. in the diastolic pressure. These variations may be interpreted as resulting from excitement produced by automobiling. A neurotic woman stated that the act of taking the blood-pressure always caused fright similar to that occasioned by the near approach of a rapidly-moving horse and carriage while crossing the street. Fright thus induced caused a systolic pressure of from 205 to 235 mm., and a pulse pressure of from 65 to 90 mm.

Physiologic conditions must be secured before making a blood-pressure determination. No patient, more especially one of a nervous temperament, should be examined immediately after arriving at the physician's office, unless a control observation is made later, and any evidence of haste or excitement should be avoided.

The first observation is made in the sitting posture, the second after reclining for a few minutes, and a third immediately after making ten vigorous bending movements, touching the fingers to the floor with each movement.

The average systolic pressure in an adult in the sitting posture, under average physiologic conditions, is from 120 to 130 mm., and the average diastolic pressure is 20 or 30 mm. less. Occasionally the normal blood-pressure of

certain individuals may be 5, 10 mm. or more, higher or lower. In recumbency, as a rule, the systolic and diastolic pressure falls 5 or 10 mm., although occasionally the systolic pressure may remain unchanged and the diastolic pressure fall 5 or 10 mm. Immediately after exercise the systolic and diastolic pressure are ordinarily 5 or 10 mm. higher than that obtained in the sitting posture. The average pulse amplitude or pulse pressure is about 20 or 30 mm. Occasionally it is desirable that the blood-pressure be determined after rapidly walking a certain known distance, or after running rapidly up and down three flights of stairs.

Valuable information as to the state of the nervous system, and especially of the vasomotor system, is obtained from blood-pressure observations based upon the foregoing considerations.

In many cases of failure of compensation of the heart the blood-pressure tends to decrease and the pulse-pressure remains approximately normal. In well-marked aortic regurgitation, where compensation is preserved, the systolic pressure is usually high, the diastolic low, and the pulse-pressure may be as much as 60 mm. or more.

When the myocardium is organically or functionally weak, there is a tendency to hypotension. When compensation has been restored in the various diseases of the heart the blood-pressure may not be materially altered. As blood-pressure is dependent not alone upon the condition of the myocardium and vessels, but also upon the calibre of the arterioles and capillaries, extreme variations in blood-pressure may be observed in the course of various forms of heart disease.

Uræmia is a cause of persistent hypertension, and systolic pressures of 180 to 315 mm. have been observed. Hypertension from this cause is usually uninfluenced by most drugs. A reduction of 20 mm. or more may be secured after

profuse sweating induced by a cabinet bath or pilocarpin. In my experience nitroglycerine has been disappointing in its effects upon most cases of hypertension, even when given in ascending doses at short intervals, and usually the reduction in pressure has been but slight and transitory with no symptomatic improvement. Hypertension in uræmia may be chiefly due to stimulation of the centric or peripheral vasomotor centres. When the nervous and muscular mechanism of the heart is normal an increase in the strength of each systole may be largely due to an increase in the intraventricular pressure secondary to vasomotor constriction. Vasomotor instability is a frequent cause of blood-pressure variations. In certain cases of acute lead poisoning, gout, intestinal toxæmia and disturbance of the chemistry of intestinal digestion, hypertension has been observed.

By far the largest number of cases of hypertension in private practice occur in nervous exhaustion, particularly in those who are neurotic or neuropathic. Comparatively insignificant causes are capable of producing in these individuals notable variations in blood-pressure, usually tending toward hypertension. In psycho-neurasthenia, readings of from 170 to 315 mm., with a pulse-pressure of from 30 to 60 mm. or more, have been observed. In chronic nervous exhaustion hypertension may persist continuously for months or years, eventually causing arterial sclerosis and left ventricular hypertrophy, which may eventually lead to mitral and cardiac insufficiency. In certain cases of nervous exhaustion with hypertension variation of 10, 15, or 20 mm. may occur within a few minutes, apparently due to vasomotor ataxia. In certain neurasthenics the blood-pressure in the sitting position is unchanged by recumbency, or the systolic pressure may rise, and the diastolic pressure may increase or decrease. In like manner exercise may produce no change in the blood-pressure as compared with that taken

in the sitting posture, and occasionally a lowering is observed.

These marked variations from the normal seem best explained by viewing the vasomotor system as inco-ordinate or ataxic. Vasomotor ataxia presents its most classical clinical picture in certain cases of well-marked menopause, many of whom present not only extreme variations, but, at times, extreme hypertension, rising to 200, 235, or even 270 mm., associated with a pulse-pressure varying between 60 and 110 mm.

Hypertension may be caused by excessive secretion of the thyroid or adrenals. In a case of advanced Addison's disease, with great destruction of the adrenals, the systolic pressure was 90 mm. and the diastolic pressure was 70 mm.

Time will not permit further discussion of this subject, nor of the treatment of abnormalities of blood-pressure; but it is obvious that the best results may be expected from removal of the cause.

THE ACTION OF CARBON DIOXIDE BATHS ON THE BLOOD-PRESSURE IN CARDIAC DISEASE.

By JOSEPH H. PRATT, M.D.

BOSTON.

(*From the Institution for Physical Therapeutics, Boston.*)

Of the physiological action of the carbon dioxide baths little is definitely known, in spite of the large number of papers that have been written on the subject and the extensive use that has been made of this form of treatment in cardiac disease during the past twenty years.

The literature abounds in impressions and speculations. With justice Huchard lamented the lack of facts and figures. At the present day there is not even an agreement among the few trained investigators who have attempted to decide by physiological observations whether the baths increase or diminish the work of the heart. At the International Congress of Physiotherapy held last March in Berlin, Otfried Müller [1] held to the more generally accepted view that the cool carbon dioxide baths are a form of exercise. "They make the heart do more work," Müller stated, "because in the first place they increase the systolic output of the heart, and, secondly, they produce a contraction of the arteries, accompanied by an elevation of the blood-pressure. They are in consequence procedures that surely increase the

work of the heart, and are not means of resting or protecting that organ." His conclusions are based on careful investigations made with sphygmomanometer, sphygmograph, plethysmograph and tachograph on the healthy subject.

On the other hand, Munk [2], in a paper prepared for the same Congress, asserted that the carbon dioxide bath lightens the work of the heart (Schonung des Herzen). It should be pointed out, however, that the experimental work which he presented did not warrant this conclusion.

It would seem as if the action of the baths on the blood-pressure would be a simple matter to decide, but even on this point writers are not in accord. Some observers claim that the blood-pressure is lowered, while a larger number state that it is increased.

Ewald [3], using Basch's sphygmomanometer, observed that the blood-pressure fell or remained unchanged, but he used only a warm bath, the temperature being 38° C. J. F. H. Broadbent [4] stated that "There was a lowering of tension in the peripheral circulation." In the opinion of Leslie Thorne [5] the pulse is decreased in tension as a general rule. Bezly Thorne [6] said "the tension is reduced." Gräupner [7] claimed there was a sinking of blood-pressure which in some cases lasted over two hours. L. Fellner [8] said that he could demonstrate a sinking of the blood-pressure during the bath. In an abstract of a paper by Balthusewiez [9], a Russian author, the statement is made that the blood-pressure falls a little after the baths. The temperature of the baths was 35° C., and the duration thirty minutes. Litten and Lenhoff [10] claim that there is a primary fall of blood-pressure and a secondary rise. None of these writers support their statement by any figures except L. Fellner, and an examination of his tables show that the blood-pressure usually rose after the cool baths.

A large majority of writers have held that the blood-

pressure is raised by the baths. Kingscote [11], Saundby [12], Leith [13], Sir T. Grainger Stewart [14], and Campbell [15], writing prior to 1900, stated that the blood-pressure is increased, but their opinion was based on palpation of the pulse. Stiftler [16] and Heineman [17] found an increase of pressure as measured by the Basch sphygmomanometer. A. Schott [18] stated that the blood-pressure after the Nauheim bath is raised 50 to 60 mm. of mercury. T. Schott [19] wrote in 1901 that the blood-pressure as measured by Basch's apparatus was raised 20, 30, 40 mm. or even more. Both of these writers content themselves with this general statement. Hensen [20] made observations on the effect of sixty-four baths which he gave to patients with cardiac insufficiency and other mild cardiac disorders. He found that the blood-pressure rose usually 20 to 30 mm. of mercury. There was an increase of blood-pressure in forty-seven out of a series of sixty-four baths. In eleven of the sixty-four baths there was no pronounced change of the blood-pressure; in eight a definite fall was noted.

Battistini and Rovere [21] gave 139 carbon dioxide baths to thirteen patients. An increase of blood-pressure was usually observed. The maximum rise was 40 mm.

Otfried Müller [22] in 1902 offered an explanation for the conflicting results of the earlier unsystematic investigations. He presented evidence to show that the changes of blood-pressure in a series of carbon dioxide baths given to a healthy individual at a temperature of 34° C. and of sixteen minutes' duration were due to the temperature of the bath, and not to the carbon dioxide or salt content. He claimed that baths above the indifferent point lowered the blood-pressure, and baths below this point raised the blood-pressure. These observations were made on one healthy individual. In addition, Müller recognized that in a patient with a weak heart a fall of blood-pressure might occur in a

cool carbon dioxide bath instead of the physiological rise. He reported a case of a man, aged 32, who had been kept in bed a long time on account of a decompensated mitral insufficiency. A carbon dioxide bath of moderate strength at a temperature of 33° C. produces a rise of 22·5 mm. Hg. during the bath, followed by a negative period of three hours' duration. Later, when the cardiac insufficiency was more severe, a second bath was given. There was now, instead of the initial rise, a fall of 12·5 mm., and the depression of the blood-pressure continued for five hours.

Müller made examinations of the blood-pressure in six cases of moderate cardiac failure. The rise in blood-pressure varied between 10 and 50 mm. Hg. According to Müller, either the high temperature of the bath or a weak heart explained the fall of the blood-pressure sometimes observed.

The following year Jakob [23], of Cudowa, whose early writings on carbon dioxide baths antedate those of Groedel or Schott, published a large series of observations made with Basch's apparatus. He used baths at a temperature of 36° or 35° C., and found that the blood-pressure was usually increased.

Müller's observations were made with the Riva Rocci manometer, as were those of Hensen, and Battistini and Rovere. O. Müller was the first investigator of this subject to use the broad armlet devised by v. Recklinghausen.

In 1903 Reisner and Grote [24] published the first systematic study of the action of the natural carbon dioxide baths at Nauheim on the blood-pressure of patients with cardiac insufficiency. They used baths at 32° to 34° C., and found the blood-pressure usually elevated. They employed the Gärtner tonometer.

One of the most complete clinical studies of the influence of carbon dioxide baths on the blood-pressure was carried out by Tiedeman and Lund [25] in Krehl's clinic. This

appeared in 1907. The observations were made on ten patients the subject of valvular disease and myocarditis.

The best contribution to this subject that I have seen in English is the paper by Swan [26], which was published last year. He showed clearly that a course of carbon dioxide brine baths had no constant effect on the blood-pressure. In only five of the eighty-one cases did he observe the action of the individual baths on the blood-pressure. In these he found that the systolic pressure was raised more frequently than it was lowered. The value of Swan's paper is impaired by the absence of any data regarding the temperature, strength, or duration of the baths. In another article he states [27] that the temperature ranged from 96° F. for the weaker baths to 85° F. for the strongest. The content of carbon dioxide varied from "3,702 cubic inches to 17,720 cubic inches." The duration was from five to fifteen minutes.

A critical examination of all this earlier work leads to the conclusion that the weight of evidence is with those who claim that the cool baths raise the blood-pressure. If the blood-pressure fell in a cool bath that would indicate, in the light of Otfried Müller's work, that the heart was weak.

In his first paper (1902) Müller said cases with severe decompensation should not be given carbon dioxide baths, as cardiac strain might result. In one case Müller attributed an attack of pulmonary œdema to the baths.

Matthes [28] says if the blood-pressure falls the use of the carbon dioxide baths should be stopped. Lommel [29] holds a similar view.

For a long time I have regarded the action of the baths on the blood-pressure as an important guide in the administration of this form of treatment. Two years ago, at the Montreal meeting of the Climatological Society, I proposed

a functional test of the heart based on the use of the carbon dioxide bath [30]. At that time I thought that if a full-strength carbon dioxide bath at 90° F. and of ten minutes' duration lowered the blood-pressure the heart was weak. It was my impression that my own clinical experience had demonstrated the value of this method in deciding whether a course of carbon dioxide baths would be beneficial or harmful.

Louis* stated many years ago that when he attempted to confirm his clinical impressions by an impartial study of carefully collected facts he invariably found that his impressions had been misleading. This has been my experience in the present investigation.

Material and Methods.—The present series of observations has extended over a period of seven years. After undressing the patients rested fifteen minutes to half an hour in the recumbent posture. For the bath a large tub was used, so that the entire body, except the head and neck, was immersed in the water. The head and shoulders were supported in a comfortable position by an air cushion. The blood-pressure determinations were made by trained assistants or by myself. A mercury manometer of the Riva Rocci type was used and the broad armlet. The diastolic pressure was determined by the auscultatory method. The blood-pressure was measured with the patient resting quietly on a couch a few minutes before entering the bath. All the observations on the same patient were usually made by

* "Now no circumstances could possibly have been more favourable to test the value of experience than those in which M. Louis was placed, yet when at the close of his labours he submitted all his facts to the unerring test of arithmetical analysis, in every instance were the *a priori* conclusions which he had formed from the recollection of his own facts found to be erroneous. This most remarkable result ought to be indelibly engraven on the mind of every observer, and inspire a doubt as to the validity, not only of the experience of others, but of what he has hitherto perhaps considered almost infallible, his own."—Introductory chapter by C. Cowan in Louis's "Pathological Researches on Phthisis," Boston, 1836, p. xxxvi.

one person. In this way the disturbing psychic factor that would have resulted from the presence of different individuals on different days was avoided. Most of the patients took ten to twenty baths, and were accustomed to the entire procedure by the time the stronger and cooler baths were taken. At the end of the bath the patient was assisted in getting out of the tub by the attendant and the skin quickly dried. The patient was not allowed to rub himself or to make any other exertion, except to walk to a rest-room, which was not more than twenty to thirty steps from the bath tub. The blood-pressure was determined with the patient in the recumbent posture a few minutes after the bath.

The baths were prepared according to the method described by Matthes [28]. The full strength bath contained 1,000 c.c. of HCl, and is designated the 100 per cent. bath. Usually an equal volume of sodium bicarbonate was used, but sometimes twice the volume. The amount in each bath is recorded in the tables.

From the large number of observations made by me or under my direction, I have selected determinations on ten different patients for each degree of temperature from 95° F. to 85° F. This includes the upper and lower limits of temperature at which the carbon dioxide baths have been generally employed in cardiac cases.

The Carbon Dioxide Bath as a Functional Test of the Heart.—Mention has been made of the case of O. Müller in which with increase of cardiac insufficiency a carbon dioxide bath of the same temperature, strength, and duration, as that which had previously produced a rise of blood-pressure caused a fall.

The observations made on the following case suggested that the action of carbon dioxide baths might be a guide in determining the functional power of the heart.

J. O., male, aged 53. Diagnosis: Chronic myocarditis; dilatation of the heart; cardiac cirrhosis; ascites.

When I saw him for the first time, on February 12, 1907, he had been ill for about eight months. Recurring ascites and nocturnal restlessness and dyspnœa on exertion were the chief symptoms. The heart was dilated and the pulse weak and irregular. There was some ascites and œdema of the legs.

TABLE I.

Date	Temperature of bath	Strength CO_2	Blood-pressure		Effect of bath on blood-pressure		Pulse	
			Before	After	Rise	Fall	Before	After
	Degrees	Per cent.						
Jan. 27	95	20	140	190	50	—	88	88
Feb. 4	92	40	160	180	20	—	60	65
,, 6	92	50	160	180	20	—	100	92
,, 8	91	60	150	175	25	—	92	84
,, 11	90	70	150	170	20	—	104	104
,, 12	90	70	155	170	15	—	100	100

Average rise of blood-pressure 25 mm. Hg.

The œdema disappeared and the other symptoms lessened. Against advice he discontinued the baths. He worked at his office from March 1 until the middle of April. Ascites and other symptoms of cardiac insufficiency suddenly recurred. The heart was markedly dilated to the right and to the left. Rest in bed, digitalis and a restricted intake of fluids produced marked improvement. On May 16 he began a second course of carbon dioxide baths. At that time there was little or no dilatation of the heart and the ascites and subcutaneous œdema had disappeared. He walked up a flight of stairs on his return from the bath without difficulty or breathlessness.

In February the carbon dioxide baths produced a marked rise of blood-pressure, while in May, as Table II shows, baths of the same strength and temperature as those given

in February produced much less of a rise. The average in the first series was 25 and in the second 3.

TABLE II.

Date	Temperature of bath	Strength CO_2	Blood-pressure		Effect of bath on blood-pressure		Pulse	
			Before	After	Rise	Fall	Before	After
	Degrees	Per cent.						
May 16	93	20	160	180	20	—	84	84
,, 18	93	40	178	184	6	—	80	80
,, 20	92	50	173	180	7	—	92	90
,, 22	91	60	190	183	—	7	96	96
,, 24	90	60	175	165	—	10	92	92
,, 27	90	50	170	180	10	—	90	90
,, 29	90	50	180	185	5	—	84	84

Average rise of blood-pressure 4 mm. Hg.

Although from his symptoms and physical examination one might have concluded that his heart was stronger in May than in February the action of the baths on the blood-pressure seemed to indicate that the heart was weaker.

Cardiac insufficiency developed after he attempted to work, and death occurred within a few months.

It is evident that the study of the action of carbon dioxide baths at different temperatures on strong and weak hearts will show whether this test is trustworthy or not. In none of the cases analysed in the following tables was there severe cardiac insufficiency, and two (C. J. and C. E.) have had no symptoms of cardiac weakness in the seven and eight years respectively that have elapsed since the baths were taken.

O. Müller and Kommerell [31] found that baths at 35° C. lowered the blood-pressure in the single healthy subject upon whom they made observations.

In der Stroth [32], working with O. Müller, found the

indifferent zone, in which blood-pressure was not affected, to lie between 32° and 34° C. in the two subjects he examined.

As Müller has observed a fall of blood-pressure with carbon dioxide baths at 34° and 35° C. with healthy individuals he has concluded that baths at these temperatures rest the heart. Table III shows that six of my ten patients had a distinct elevation of blood-pressure when a bath at 35° C. was given. The content in carbon dioxide was so small as to be almost negligible. If a rise of pressure after a plain water bath or a carbon dioxide bath is evidence of increased work, then, contrary to what one would expect from Müller's investigation, in the majority of my patients the bath at 35° C. increased rather than lightened the load of the heart.

To ascertain whether or not these 10 cases were exceptional ones I have collected observations on a total of 35 baths on different individuals at 35° C. The duration varied from seven to ten minutes. There was a rise in 23, a fall in 6, and no change in 6.

In one patient (E. J.) baths above 35° C. were given, with the following results :--

Bath at 98° *F.* 36·7° *C.*

	Strength. Per cent.	Duration. Minutes	Blood-pressure			Pulse		Amount of sodium bicarbonate
			Before	After	Fall	Before	After	
May 16...	10	7	215	170	45	76	78	100

Bath at 96° *F.* 36·6° *C.*

	Strength. Per cent.	Duration. Minutes	Blood-pressure			Pulse		Amount of sodium bicarbonate
			Before	After	Fall	Before	After	
May 18...	20	9	210	160	50	86	86	200

With this patient the indifferent point was between 95° F. and 96° F. In a series of six baths given to her at temperatures below 96° F. there was always a rise of blood-pressure.

TABLE III.

Bath at 95° F. 35° C.

Name	Strength of bath	Duration	Blood-pressure					Pulse				Cubic centimetres of $HNaCO_3$*
			Before	After	Rise	Fall	No change	Before	After	Rise	Fall	
	Per cent.	Minutes										
(1) B. B.	30	7	95	100	5	—	—	67	64	—	3	600
(2) A. B.	20	8	120	120	—	—	0	84	68	—	16	400
(3) E. D.	10	7	180	185	5	—	—	96	88	—	8	100
(4) C. E.	10	7	95	115	20	—	—	92	82	—	10	200
(5) H. F.	30	7	130	115	—	15	—	84	80	—	4	600
(6) W. J.	10	7	155	155	—	—	0	72	70	—	2	200
(7) C. J.	30	7	105	115	10	—	—	98	115	17	—	300
(8) E. J.	10	7	155	180	25	—	—	80	82	2	—	100
(9) W. M.	30	7	142	135	—	7	—	88	88	—	—	600
(10) J. O.	20	7	140	190	50	—	—	88	88	—	—	400

In 6 cases rise of blood-pressure.
In 2 cases fall of blood-pressure.
In 2 cases no change.

In 2 cases rise of pulse-rate.
In 6 cases fall of pulse-rate.
In 2 cases no change.

* For convenience in preparing the baths, sodium bicarbonate was measured by volume. 1,000 c.c. weigh 1,250 grm.

TABLE IV.

Bath at 94° F. 34·4° C.

Name	Strength of bath	Duration	Blood-pressure					Pulse				Cubic centimetres of $HNaCO_3$
			Before	After	Rise	Fall	No change	Before	After	Rise	Fall	
	Per cent.	Minutes										
(1) B. B.	40	8	105	120	15	—	—	64	64	—	—	800
(2) A. B.	30	10	100	90	—	10	—	82	84	2	—	600
(3) E. D.	20	8	170	185	15	—	—	84	72	—	12	200
(4) C. E.	20	8	100	100	—	—	0	66	70	4	—	400
(5) H. E.	30	9	110	115	5	—	—	80	72	—	8	600
(6) W. J.	20	8	145	155	10	—	—	74	72	—	2	400
(7) C. J.	40	8	110	115	5	—	—	82	78	—	4	400
(8) J. C.	30	8	155	145	10	—	—	80	76	—	4	400
(9) H. R.	20	8	135	145	10	—	—	80	84	4	—	400
(10) W.M.	50	8	120	125	5	—	—	84	84	—	—	1,000

In 8 cases rise of blood-pressure.
In 1 case fall of blood-pressure.
In 1 case no change.

In 3 cases rise of pulse-rate.
In 5 cases fall of pulse-rate.
In 2 cases no change.

In my series the one patient who showed a drop at 94° F. had no change in the blood-pressure at 95° F., while the two patients who showed a drop at 95° F. had a rise when the temperature was 1° lower.

TABLE V.

Bath at 93° F. 33·9° C.

Name	Strength of bath	Duration	Blood-pressure					Pulse				Cubic centimetres of $HNaCO_3$
			Before	After	Rise	Fall	No change	Before	After	Rise	Fall	
	Per cent.	Minutes										
(1) B. B.	50	10	110	120	10	—	—	66	68	2	—	1,000
(2) E. D.	30	9	165	175	10	—	—	84	78	—	6	300
(3) C. E.	30	9	100	110	10	—	—	82	80	—	2	600
(4) H. F.	20	9	130	110	—	20	—	108	100	—	8	1,000
(5) W. J.	40	9	140	140	—	—	0	80	76	—	4	800
(6) C. J.	50	10	110	110	—	—	0	82	78	—	4	600
(7) J. O.	20	7	160	180	20	—	—	84	84	—	—	400
(8) J. M.	30	9	195	200	5	—	—	104	88	—	16	600
(9) W. M.	30	10	125	130	5	—	—	92	88	—	4	300
(10) H. R.	30	9	125	120	—	5	—	84	88	4	—	600

In 6 cases rise of blood-pressure.
In 2 cases fall of blood-pressure.
In 2 cases no change.

In 2 cases rise of pulse-rate.
In 7 cases fall of pulse-rate.
In 1 case no change.

TABLE VI.

Bath at 92° F. 33·3° C.

Name	Strength of bath	Duration	Blood-pressure					Pulse				Cubic centimetre of $HNaCO_3$
			Before	After	Rise	Fall	No change	Before	After	Rise	Fall	
	Per cent.	Minutes										
(1) B. B.	60	11	110	125	15	—	—	64	64	—	—	1,200
(2) A. B.	40	12	90	90	—	—	0	62	72	10	—	800
(3) E. D.	40	10	160	170	10	—	—	84	76	—	8	400
(4) C. E.	40	10	105	110	5	—	—	80	80	—	—	800
(5) W. J.	50	10	130	150	20	—	—	66	70	4	—	1,000
(6) E. J.	50	15	190	195	5	—	—	98	88	—	10	500
(7) J. O.	50	10	173	180	7	—	—	92	90	—	2	1,000
(8) J. M.	40	10	180	200	20	—	—	81	84	3	—	800
(9) J. C.	50	10	160	170	10	—	—	68	80	12	—	1,000
(10) H. R.	40	10	130	135	5	—	—	92	88	—	4	800

In 9 cases rise of blood-pressure.
In 1 case no change.

In 4 cases rise of pulse-rate.
In 4 cases fall of pulse-rate.
In 2 cases no change.

TABLE VII.

Bath at 91° *F.* 32·8° *C.*

Name	Strength of bath	Duration	Blood-pressure					Pulse				Cubic centimetres of $HNaCO_3$
			Before	After	Rise	Fall	No change	Before	After	Rise	Fall	
	Per cent.	Minutes										
(1) B. B.	70	12	110	120	10	—	—	60	66	6	—	1,400
(2) E. D.	50	11	160	175	15	—	—	88	84	—	4	500
(3) C. E.	50	11	90	110	20	—	—	94	90	—	4	1,000
(4) H. F.	30	15	140	115	—	25	—	108	96	—	12	300
(5) J. C.	50	11	135	142	7	—	—	80	76	—	4	1,000
(6) E. J.	60	16	160	190	30	—	—	96	72	—	24	600
(7) J. O.	60	11	190	183	—	7	—	96	96	—	—	1,200
(8) W. M.	100	10	122	136	14	—	—	82	84	2	—	1,000
(9) J. M.	50	11	158	170	12	—	—	78	70	—	8	1,000
(10) H. R.	50	11	125	145	20	—	—	84	80	—	4	1,000

In 8 cases rise of blood-pressure.
In 2 cases fall of blood-pressure.

In 2 cases rise of pulse-rate.
In 7 cases fall of pulse-rate.
In 1 case no change.

The patient H. F., who showed a marked fall also had a depression of the blood-pressure at 92° F. It should be pointed out that she had a weak heart and a bath given at 95° F. produced a drop of blood-pressure, as shown in Table III. Note that her heart action was more rapid than

TABLE VIII.

Bath at 90° *F.* 32·2° *C.*

Name	Strength of bath	Duration	Blood-pressure					Pulse				Cubic centimetres of $HNaCO_3$
			Before	After	Rise	Fall	No change	Before	After	Rise	Fall	
	Per cent.	Minutes										
(1) B. B.	80	13	115	125	10	—	—	68	68	—	—	1,600
(2) A. B.	50	14	90	100	10	—	—	68	68	—	—	1,000
(3) E. D.	60	12	160	160	—	—	0	82	80	—	2	600
(4) C. E.	60	12	90	90	—	—	0	64	64	—	—	1,200
(5) H. F.	70	19	115	120	5	—	—	84	88	4	—	700
(6) W. J.	70	12	140	170	30	—	—	76	72	—	4	1,400
(7) C. J.	80	15	105	95	—	10	—	88	82	—	5	800
(8) E. J.	90	18	175	200	25	—	—	66	62	—	4	700
(9) W. M.	60	13	105	110	5	—	—	88	76	—	12	600
(10) J. O.	60	12	175	165	—	10	—	92	92	—	—	1,200

In 6 cases rise of blood-pressure.
In 2 cases fall of blood-pressure.
In 2 cases no change.

In 1 case rise of pulse-rate.
In 5 cases fall of pulse-rate.
In 4 cases no change.

that of any other of the patients, who took the bath at 91° F.

The patient C. J. probably had a normal heart. He has had no cardiac symptoms in the seven years that have passed since this bath was taken. This patient had no rise of blood-pressure after a bath at 93° F., and only an elevation of 5 mm. at 94° F. When cooler baths were given (88° F. and 87° F.) the blood-pressure rose 15 mm. Of 25 patients who took a carbon dioxide bath at 90° F., in a series I have analysed, the blood-pressure was elevated in 21, there was a fall in 1, and no change in 2. C. J. was the one in this series of 25 patients who had the fall of blood-pressure. The average rise in the 22 patients whose blood-pressure was increased amounted to 17 mm. of Hg. This series at 90° F. showed conclusively that a patient with a strong heart may have a fall of blood-pressure in a cool bath, while patients with distinct loss of cardiac power may have a rise of blood-pressure.

TABLE IX.

Bath at 89° F. 31·7° C.

Name	Strength of bath	Duration	Blood-pressure					Pulse				Cubic centimetres of $HNaCO_3$
			Before	After	Rise	Fall	No change	Before	After	Rise	Fall	
	Per cent.	Minutes										
(1) B. B.	90	14	110	125	15	—	—	64	68	4	—	1,800
(2) E. D.	90	13	165	175	10	—	—	76	74	—	2	700
(3) C. E.	60	15	100	110	10	—	—	84	76	—	8	1,200
(4) H. E.	40	13	100	100	—	—	0	88	58	—	20	800
(5) H. F.	90	20	125	120	—	5	—	76	74	—	2	900
(6) W. J.	80	10	145	160	15	—	—	74	71	—	3	1,600
(7) W. M.	70	14	95	100	5	—	0	95	88	—	7	700
(8) J. O.	40	13	170	170	—	—	—	90	90	—	—	800
(9) J. C.	60	12	140	152	12	—	—	72	84	12	—	1,200
(10) J. M.	70	13	160	180	20	—	—	70	72	2	—	1,400

In 7 cases rise of blood-pressure.
In 1 case fall of blood-pressure.
In 2 cases no change.

In 4 cases rise of pulse-rate.
In 5 cases fall of pulse-rate.
In 1 case no change.

The only patient whose blood-pressure fell in the bath given at 89° F. had a weak heart.

TABLE X.

Bath at 88° F. 31·1° C.

Name	Strength of bath	Duration	Blood-pressure					Pulse				Cubic centimetres of $HNaCO_3$
			Before	After	Rise	Fall	No change	Before	After	Rise	Fall	
	Per cent.	Minutes										
(1) B. B.	100	15	115	130	15	—	—	60	64	4	—	2,000
(2) A. B.	70	17	100	114	14	—	—	72	68	—	4	1,400
(3) E. D.	80	14	200	165	—	35	—	80	76	—	4	800
(4) C. E.	70	16	95	110	15	—	—	84	82	—	2	1,400
(5) H. E.	40	15	120	100	—	20	—	88	84	—	4	800
(6) H. F.	90	18	110	115	5	—	—	76	74	—	2	900
(7) W. J.	90	10	130	165	35	—	—	70	70	—	—	1,800
(8) C. J.	100	—	80	95	15	—	—	76	72	—	4	1,000
(9) E. J.	100	20	175	190	15	—	—	80	78	—	2	1,000
(10) J. O.	40	14	180	175	—	5	—	100	96	—	4	800

In 7 cases rise of blood-pressure.
In 3 cases fall of blood-pressure.

In 1 case rise of pulse-rate.
In 8 cases fall of pulse-rate.
In 1 case no change.

In the two cases in which there was a marked fall of blood-pressure in the bath at 88° F. there was no shortness of breath on exertion. One of these, E. D., had chronic interstitial nephritis without signs of cardiac insufficiency. H. E. was subject to attacks of palpitation of the heart.

TABLE XI.

Bath at 87° F. 30·5° C.

Name	Strength of bath	Duration	Blood-pressure					Pulse				Cubic centimetres of $HNaCO_3$
			Before	After	Rise	Fall	No change	Before	After	Rise	Fall	
	Per cent.	Minutes										
(1) B. B.	100	16	110	125	15	—	—	64	67	3	—	2,000
(2) A. B.	80	17	112	112	—	—	0	62	60	—	2	1,600
(3) E. D.	90	15	185	195	10	—	—	80	70	—	10	900
(4) C. E.	80	17	105	125	20	—	—	84	80	—	4	1,800
(5) H. E.	40	17	100	105	5	—	—	84	66	—	18	800
(6) W. J.	90	13	145	160	15	—	—	75	70	—	5	1,800
(7) C. J.	100	20	110	125	15	—	—	68	68	—	—	1,000
(8) J. O.	40	15	170	170	—	—	0	96	92	—	4	800
(9) J. C.	60	12	150	148	—	2	—	76	72	—	4	1,200
(10) H. C.	100	16	145	150	5	—	—	72	72	—	—	2,000

In 7 cases rise of blood-pressure.
In 1 case fall of blood-pressure.
In 2 cases no change.

In 1 case rise of pulse-rate.
In 7 cases fall of pulse-rate.
In 2 cases no change.

At the temperature of 87° F. the drop of blood-pressure in the single case in which it was observed was so slight that this case might be as properly classed with the two in which the blood-pressure was unchanged.

TABLE XII.

Bath at 85° F. 30° C.

Name.	Strength of bath	Duration	Blood-pressure					Pulse				Cubic centimetres of $HNaCO_3$
			Before	After	Rise	Fall	No change	Before	After	Rise	Fall	
	Per cent.	Minutes										
(1) B. B.	100	17	110	125	15	—	—	64	64	—	—	2,000
(2) A. B.	90	20	140	120	—	20	—	62	72	10	—	1,800
(3) E. D.	100	16	185	210	25	—	—	80	78	—	2	1,000
(4) C. E.	90	18	115	100	—	15	—	80	78	—	2	1,800
(5) H. E.	50	19	110	112	2	—	—	84	66	—	18	1,000
(6) H. F.	100	20	120	115	—	5	—	80	84	4		1,000
(7) E. J.	100	20	175	195	20	—	—	78	72	—	6	1,000
(8) J. O.	50	15	150	165	15	—	—	100	100	—	—	1,000
(9) H. C.	100	17	130	155	25	—	—	80	76	—	4	2,000
(10) H. D.	100	16	110	135	25	—	—	72	72	—	—	2,000

In 7 cases rise of blood-pressure.
In 3 cases fall of blood-pressure.

In 2 cases rise of pulse-rate.
In 5 cases fall of pulse-rate.
In 3 cases no change.

TABLE XIII.

Bath at 85° F. 29·4° C.

Name	Strength of bath	Duration	Blood-pressure					Pulse				Cubic centimetres of $HNaCO_3$
			Before	After	Rise	Fall	No change	Before	After	Rise	Fall	
	Per cent.	Minutes										
(1) B. B.	100	18	115	125	10	—	—	61	62	1	—	2,000
(2) A. B.	70	20	140	110	—	30	—	60	64	4	—	1,400
(3) E. D.	100	17	160	185	25	—	—	80	76	—	4	1,000
(4) C. E.	100	19	100	100	—	—	0	96	80	—	16	2,000
(5) H. E.	50	20	105	105	—	—	0	76	68	—	8	1,000
(6) H. F.	100	20	115	115	—	—	0	72	74	2		1,000
(7) H. C.	100	18	140	175	35	—	—	76	72	—	4	2,000
(8) F. E.	100	20	130	130		—	0	70	70	—		1,000
(9) H. D.	100	17	114	120	6	—	—	80	76		4	2,000
(10) H. R.	100	16	125	120	—	5	—	72	76	4		—

In 4 cases rise of blood-pressure.
In 2 cases fall of blood-pressure.
In 4 cases no change.

In 4 cases rise of pulse-rate.
In 5 cases fall of pulse-rate.
In 1 case no change.

Neither of the two cases in which the blood-pressure fell in the bath at 87° F. showed a fall at this lower temperature. On the other hand, the blood-pressure in A. B. and C. E. fell at 86° F., but not at 87° F.

Bath patients whose blood-pressure fell in the coldest bath of the series were able to exercise without breathlessness at the time the bath was taken.

TABLE XIV.—ACTION OF CARBON DIOXIDE BATHS AT DIFFERENT TEMPERATURES ON THE BLOOD-PRESSURE OF THE SAME INDIVIDUALS

Case	Name	Temperature of baths										
		95°	94°	93°	92°	91°	90°	89°	88°	87°	86°	85°
1	B. B.	5	15	10	15	10	10	15	15	15	15	10
2	A. B.	0	−10	—	0	–	10	—	14	0	−20	−30
3	E. D.	5	15	10	10	15	0	10	−35	10	25	25
4	C. E.	20	0	10	5	20	0	10	15	20	−15	0
5	H. E.	−15	5	—	—	—	—	0	−20	5	2	0
6	H. F.	−20	—	−20	—	−25	5	−5	5	—	−5	0
7	H. C.	—	—	—	—	—	—	—	—	5	25	35
8	F. E.	—	—	—	—	—	—	—	—	—	—	0
9	H. D.	5	—	—	—	—	—	—	—	—	25	6
10	H. R.	—	10	−5	5	20	—	—	—	—	—	−5
11	E. J.	—	—	—	5	30	25	—	15	—	20	—
12	J. O.	—	—	20	7	−7	−10	0	−5	0	−5	—
13	W. J.	0	10	0	20	—	30	15	35	15	—	—
14	C. J.	10	5	0	—	—	−10	—	15	15	—	—
15	W. M.	−7	5	5	—	14	5	5	—	—	—	—
16	J. C.	—	−10	—	10	7	—	12	—	−2	—	—
17	J. M.	—	—	5	20	12	—	20	—	—	—	—

Only five of the patients in the series of baths analysed in Table XIV showed a rise of blood-pressure after every bath (B. B., H. C., H. D., E. J., J. M.). The effect of the baths of varying strengths and temperatures was remarkably uniform in the case of B. B. The rise of pressure was never less than 5 nor more than 15. During a period of three years this patient took 70 carbon dioxide baths. In 65 the blood-pressure rose, in 3 it fell, and in 2 it remained unchanged. The patient H. C. took in all 14 baths. There

was a rise of blood-pressure after 13 of these and a fall after one.

Two patients exhibited a fall of blood-pressure after four of the baths in the present series, and both had serious disease of the myocardium and died within the following year. J. M., on the other hand, had an elevation of the blood-pressure after 19 of the 20 baths which he took. This was not evidence of a strong heart, as it was necessary to give digitalis while he was taking the course of baths. A few months later severe cardiac insufficiency developed.

Sometimes a bath will produce a fall of blood-pressure one day and the next day a bath of the same temperature, gas content, and duration will be followed by a rise of blood-pressure, or *vice versâ*. For example, E. J. took a carbon dioxide bath of full strength on June 21 at 88° F. and of twenty minutes' duration, after which the blood-pressure fell from 110 to 100 mm. On the following day this bath produced an elevation of the blood-pressure from 80 to 95 mm. of mercury. It will be noted that the maximum fall of pressure, 35 mm. of Hg., occurred in a patient who had a rise of blood-pressure two days previously and also two days later after baths of nearly the same strength. Similar inexplicable drops of pressure are found elsewhere in Table XIV. The patient C. E. shows a distinct rise after a bath at 87° F. and a well-marked fall two days later after a bath at 86° F.

Similar observations have been made by earlier investigators. Reisner and Grote studied the action of the natural carbon dioxide baths of Nauheim on the blood-pressure. One day a bath at 33° C. produced a rise of 38 mm. of mercury in a patient with an abnormally low blood-pressure. Three days later a similar bath produced no alteration of the initial blood-pressure.

Tiedeman and Lund report a case in which a bath at

31° C., containing 5 kg. of Nauheim salt, 900 grm. of sodium bicarbonate, and hydrochloric acid, produced a fall of blood-pressure from 122 to 120. The next day a bath at the same temperature and of the same strength raised the blood-pressure from 140 to 150 mm.

TABLE XV.—ACTION OF CARBON DIOXIDE BATHS ON THE BLOOD-PRESSURE OF TEN PATIENTS AT TEMPERATURES RANGING FROM 95° F. TO 85° F.

Temperature		Average strength in CO_2	Average duration	Blood-pressure				Pulse	
				Number of cases increased	Number of cases decreased	Number of cases unchanged	Average rise mm. Hg.	Number of cases increased	Number of cases decreased
Degrees F.	Degrees C.	Per cent.	Minutes						
95	35	20	7	6	2	2	9·3	2	6
94	34·4	30	8	8	1	1	6·5	3	5
93	33·9	33	9	6	2	2	3·5	2	7
92	33·3	46	11	9	0	1	9·7	4	4
91	32·8	57	12	8	2	0	9·6	2	7
90	32·2	68	14	6	2	2	6·5	1	5
89	31·7	69	14	7	1	2	8·2	4	5
88	31·1	74	15	7	3	0	5·4	1	8
87	30·5	78	16	7	1	2	8·3	1	7
86	30·0	88	18	7	3	0	8·7	2	5
85	29·4	92	19	4	2	4	4·1	4	5

The average rise was calculated by subtracting the total fall of blood-pressure from the total rise in the ten cases at each different temperature and dividing by ten.

It is clearly seen from this table that the carbon dioxide baths at all temperatures at which they have been used in the treatment of heart disease tend to raise the blood-pressure. If this rise of peripheral blood-pressure is an indication that the heart is doing more work, then the baths at all temperatures from 35° C. to 29·4° C. usually increase the work of the heart.

The results of this analysis contradict the recent statement of Groedel [33] that "carbon dioxide baths at tem-

perature between 35° C. and 28° C. lessen the activity of the heart and protect the heart muscle."

The greatest average rise was at 91° F. and 92° F., while the coldest bath at 85° F. produced a slighter rise of blood-pressure than that of any other temperature except 93° F. It should be noted that only 1° F. separates the temperature of the bath at which the greatest and the least rise of blood-pressure occurred. This makes one suspect that another selection of cases might show more of a rise at 93° F.; and this I found to be true. I selected at random observations on four other patients who had taken baths at 93° F. and added them to the series of ten baths analysed above. The average rise in this enlarged set was 10·9 mm.—a higher figure than that found at any temperature in the large series recorded in Table XV.

It will be seen by referring to Table XV that a rise of blood-pressure occurred in at least 60 per cent. of the cases at every degree of temperature from 86° F. to 95° F., but at 85° F. only 40 per cent. showed an increase.

Table XV also shows that there is no direct relation between the strength of the bath in carbon dioxide gas or the duration of the bath and the amount of elevation of the blood-pressure.

Action of Carbon Dioxide Baths on the Pulse.—The statement has been repeatedly made that these baths lower the pulse. In this series of 110 baths it is shown in Table XV that the pulse-rate was increased 26 times and lowered 64 times. There was no direct relation between the temperature, duration or strength of the bath and the reduction of the heart-rate. A short weak bath at 95° F. produced a fall of the pulse-rate in 60 per cent. of the cases, while a strong bath of long duration at 85° F. was followed by a lowered pulse-rate in only 50 per cent. of the cases.

No clear connection seems to exist between the action

of the baths on the blood-pressure and the pulse. Usually, either a rise or fall of blood-pressure is accompanied by a fall in the pulse-rate. This was noted 45 times after the 76 baths in which the blood-pressure was elevated. In 18 of the remainder the pulse was accelerated, and in 13 it was unchanged. In 10 instances out of the 18 in which the bath produced a fall of blood-pressure the pulse-rate also fell. The fall of blood-pressure was five times accompanied by an increase of the pulse. After three baths in which the blood-pressure fell there was no change in the pulse-rate.

The patient B. B. had a rise of blood-pressure in each of the eleven baths, but the pulse fell only once, and that after the warmest bath of the series. The rate was increased after eight baths and remained stationary after three.

The pulse-rate of A. B. at a temperature of 95° F. fell 16, but the blood-pressure remained unchanged. In the case of A. J. after a bath at the same temperature the pulse was increased 17 beats and the blood-pressure rose 10 mm. The patient C. E. had a fall in the pulse-rate of 16 after a bath at 85° F., but the blood-pressure was unaltered.

Of the six out of ten patients who had a lowered pulse-rate after a bath at 95° F. the average fall was 7, and the average drop after a bath at 85° F. was also 7.

Abstracts of the Clinical Records.

Case 1.—B. B., male, aged 51 in 1906; leather manufacturer. Referred by Dr. J. H. Musser, of Philadelphia.

Diagnosis.—Coronary sclerosis. Strong and well since childhood except for chronic diarrhœa in 1904 and 1905. At times pulse irregular. Slight discomfort over præcordia occasionally when walking up steep hills. Strongly built, stout, healthy-looking man. Heart found enlarged on percussion and on orthodiagraphic examination. A soft systolic bruit replaced the first sound at the apex. First sound not audible in pulmonic or aortic areas. Took the carbon

dioxide baths in 1906 and 1907. Developed heart-block in September, 1907; pulse fell as low as 31. The heart-block persisted about four months. He has had subsequent attacks. Living and at work in 1913.

Case 2.—A. B., female, aged 46 in 1907. Referred by Dr. W. R. Steiner, of Hartford.

Diagnosis.—Mitral stenosis, auricular fibrillation. Shortness of breath on exertion since 1901. In September, 1906, walking on the level began to produce dyspnœa. Dilatation of the heart detected in December, 1906. In February, 1907, a few weeks before taking the baths walking about the house produced palpitation. Never had rheumatic fever. Stout woman of medium height. Cardiac dulness increased to the left. Thrill and diastolic murmur sometimes present at the apex. Cardiac action very irregular. Pulse weak. She made rapid and marked improvement while taking the course of carbon dioxide baths. She has had attacks of cardiac insufficiency since then, but is still able to lead a fairly active life (1913).

Case 3.—E. D., male, aged 56 in 1906; clerk. Referred by Dr. J. B. Briggs, jun., of Washington.

Diagnosis.—Chronic interstitial nephritis; diabetes. Diabetes discovered in 1905. Patient on a regulated diet; had no sugar in the urine when the baths were taken in 1906. He was perfectly well at that time. No enlargement of the heart detected on percussion. Sounds clear. Second aortic sound somewhat ringing. Pulse regular. Radial artery not thickened. Urine contained a slight trace of albumin and a few hyaline casts. He was under observation nearly two years and never complained of breathlessness on exertion or other symptoms of cardiac insufficiency. He died suddenly in December, 1907. Autopsy showed a dissecting aneurism of the aorta which had ruptured.

Case 4.—C. E., male, aged 50 in 1906; shoe manufacturer.

Diagnosis.—Coronary sclerosis; neurasthenia. Well and vigorous until 1906 when he developed insomnia as a result of worry. His pulse became irregular (extra-systoles) at that time. He never had breathlessness on exertion or pain in the chest. A tall, large-framed, stout man. Peripheral arteries not sclerosed. Heart slightly enlarged on percussion. Sounds clear. Last seen in February, 1913. He

has had no shortness of breath or other symptoms of cardiac weakness since taking the carbon dioxide baths in 1906. The arteries were not sclerosed. The heart was enlarged (orthodiagram). Heart sounds of normal quality and loudness. Numerous extra-systoles. Blood-pressure now elevated; maximum 172, minimum 120.

Case 5.—H. E., female, aged 40 in 1907.

Diagnosis.—Palpitation of the heart, possibly paroxysmal, auricular flutter, mistaken for cardiac neurosis. Attacks of cardiac palpitation accompanied by a throbbing sensation in the epigastrium since 1897. Longest interval between seizures three months. They gradually increased in length and frequency. If she walked during an attack felt weak and breathless. Pulse often normal during paroxysms, but sometimes weak. Pulse usually not accelerated, and regular. In later attacks pulse was as high as 140. No shortness of breath on exertion except during seizures. Heart not enlarged (orthodiagram and percussion). Systolic bruit at apex. Radial arteries not palpable. Several attacks of palpitation while taking the carbon dioxide baths in June, 1907. An attack of palpitation began during the last of August, 1907. It continued until her death on September 12. At the autopsy both auricles were found greatly dilated. There was chronic passive congestion of the liver, spleen, and kidneys; coronary endarteritis and chronic aortic, mitral, and tricuspid endocarditis, producing tricuspid and mitral stenosis.

Case 6.—H. F., female, aged 15 in 1905. Referred by Dr. H. W. Broughton, of Jamaica Plain.

Diagnosis.—Mitral insufficiency. In the spring of 1904 she was very sick with an attack of endo-, myo-, and pericarditis (general carditis). The left border of the heart was in the axilla. Fever lasted six weeks. After her recovery she had no shortness of breath, and there was no subcutaneous œdema. At the time she took the baths, in March and April, 1905, the area of cardiac dulness was slightly enlarged to the left. A loud systolic murmur was audible over the entire præcordia, with maximum intensity at the mitral area. In June, 1905, she had rheumatic fever. This was followed by myocardial involvement. The heart in September, 1905, was found dilated to the left. She died in July, 1906. There was œdema of high degree toward the end of life.

Case 7.—H. C., male, aged 50 in 1909. Referred by Dr. C. N. Cutler, of Chelsea.

Diagnosis.—Coronary sclerosis; neurasthenia. After the Chelsea fire he worked very hard. He became nervous and complained of weakness. Three attacks of severe pain over the heart, each attack lasting a few minutes. The first of these occurred in 1905. A slenderly built, poorly nourished man. He seemed nervous and apprehensive. Arcus senilis. Cardiac impulse forcible. Left border 2 cm. to the left of the nipple line. First sound impure at mitral area. A systolic murmur audible along the left sternal border and at the base. Brachial and radial arteries distinctly sclerosed. Pulse regular. Blood-pressure (Erlanger apparatus): maximum 170, minimum 85. Urine free from sugar and albumin. No casts.

Case 8.—F. E., female, aged 29 in 1907. Referred by Dr. S. Richmond.

Diagnosis.—Neurasthenia. Never had rheumatism. Since the birth of her first child, in 1902, has had headache almost constantly. In 1904 dull pain about the heart. She was somewhat breathless at that time on climbing stairs. Dyspnœa had not increased when she took the baths in 1907. Urine normal in the spring of 1907. Well-nourished, healthy-looking woman. Cardiac impulse not seen or felt. Left border of heart within mid-clavicular line. Absolute cardiac dulness begins just to the left of the median line and is not over 6 cm. wide. At the mitral area a faint systolic murmur accompanied the first sound. The second pulmonic markedly accentuated. Pulse regular.

Case 9.—H. D., male, aged 43 in 1907. Referred by Dr. J. H. Averill, of Brockton. Leather buyer.

Diagnosis.—Cardiac weakness following acute pericarditis and myocarditis. Several attacks of tonsillitis between 1897 and 1907. In March, 1907, had tonsillitis, followed by rheumatic fever. This was complicated by acute pericarditis. Dyspnœa for two months and cardiac arrhythmia. Slight fever until August. Examination in September, 1907, before beginning course of carbon dioxide baths. Well-built, fairly well nourished man. Temperature 98·6° F. Apex beat not visible or palpable. Cardiac dulness increased to the left, absolute cardiac dulness extending 1 cm. beyond the left nipple line. No murmurs. Pulse slow, regular,

and of good volume. On October 6 he foolishly made a considerable muscular effort. This produced some shortness of breath. After this he was more careful not to exert himself and improvement was continuous. In December tonsils were removed by Dr. Goodale. No return of cardiac symptoms in the period from December, 1907, to 1913.

Case 10.—H. R., male, aged 41 in 1909; shoe manufacturer.

Diagnosis.—Mitral stenosis; cardiac insufficiency. No history of rheumatic fever, but about twenty attacks of suppurative tonsillitis prior to 1902. In 1907 he began to be short of breath on exertion. This increased in July, 1909. No œdema. Examination July 21, 1909. Short, very stout man. Cardiac impulse not seen or felt. No thrill. Left border of dulness just outside the nipple line. Presystolic murmur at mitral area, ending in a loud, sharp first sound. Heart sounds feeble, except at apex. Heart action irregular. Radial arteries thickened. In July, after a business trip to the South, heart found dilated. In August he remained in bed two weeks and took digitalis. After this he walked a mile or more daily without shortness of breath, for the first time in a year. Took the carbon dioxide baths during September, 1909. He remained entirely free from dyspnœa until March, 1910, when he had difficulty in breathing when he walked rapidly. In October, 1910, the cardiac insufficiency became more marked, subcutaneous œdema developed and he died in March, 1911.

Case 11.—E. J., female, aged 66 in 1905. Referred by Dr. E. B. Cahill.

Diagnosis.—Chronic interstitial nephritis and cardiac insufficiency. First noticed shortness of breath on exertion about 1895. It gradually increased. Since October, 1904, has walked very little out of doors. A short, rather stout, small-framed woman. The cardiac apex was palpable in the sixth interspace outside the nipple line and 13 cm. from the median line. Both second sounds weak at the base. Pulse irregular and intermittent. Urine contained a slight trace of albumin and a few hyaline casts. The baths and graduated exercise were followed by marked improvement. She was able to walk half a mile and to ascend two flights of stairs without breathlessness. In April, 1906, dyspnœa on exertion

returned. A loud systolic murmur was present at the mitral area. Both the aortic and pulmonary second sounds were loud and clear. The radial artery was distinctly ribbed. Pulse irregular. It was necessary to interrupt the second course of carbon dioxide baths and give digitalis, which caused the shortness of breath to disappear. In June, 1907, she had marked cardiac insufficiency and chronic uræmia. She died of uræmia on June 25, 1907.

Case 12.—J. O., male, aged 53 in 1907; clerk. Referred by Dr. Samuel Crowell, of Dorchester.

Diagnosis.—Chronic myocarditis. Cardiac cirrhosis; dilatation of the heart; ascites. No history of rheumatism. Took alcohol freely for years. In 1903 or 1904 began to be short of breath on exertion. In the summer of 1906 ascites developed. Nocturnal restlessness began in September, 1906. *Status præsens,* February 12, 1907. Large-framed, somewhat emaciated man. Abdomen distended. Great enlargement of the heart. Systolic murmur at apex. Radial pulse weak and irregular. Liver plainly palpable, several centimetres below costal margin. The edge is rounded and hard. Ascites. The legs are swollen and œdematous.

Further details of this case are given in the earlier part of the paper.

Case 13.—W. J., male, aged 65 in 1907; college professor. Referred by Dr. J. J. Putnam.

Diagnosis.—Angina pectoris; coronary sclerosis. From 1898 to 1900 frequently had pain behind the sternum on slight exertion. No cardiac sensation for six years prior to December, 1906. Then the "inhibition of breathing" returned. Never had sharp pain or a sense of great pressure. Pain never radiated. No dyspnœa on exertion. Slenderly built, rather poorly nourished man. Heart normal on physical examination. Carbon dioxide baths taken in the spring of 1907. He died of heart disease in 1910.

Case 14.—C. J., male, aged 48 in 1905; physician. Never able to climb steep hills without more effort than was natural. While in college his heart was examined one day after exercising, and he was told that it was not normal. Whenever he exerted himself markedly he became breathless. This difficulty of breathing under physical stress had been present for years and had not increased. Well-built

man of medium height. Subcutaneous fat abundant. Percussion outline of heart increased. No murmurs. Pulse slow and regular. In 1910 cardiac outline normal. Heart sounds clear and of good quality. Blood-pressure 130. The carbon dioxide baths were taken in 1905. The increased area of cardiac dulness was probably due to fat in the thoracic wall. No cardiac symptoms had developed up to May, 1913.

Case 15.—W. M., male, aged 66 in 1911; book publisher.

Diagnosis.—Coronary sclerosis. On walking rapidly for a short distance he had a "cramp-like" pain across the front of his chest, attended with a sense of suffocation. For two years a sense of oppression behind the sternum whenever he walked up the slight elevation which leads to the Back Bay Station. He first had this pain in 1909 when climbing hills. No breathlessness on exertion. Apex beat not seen or felt. Cardiac dulness not definitely increased. The first sound is somewhat muffled at the apex, and it is weak at the pulmonary area. Neither sound is audible in the second interspace at the right of the sternum. The pulmonary second sound is very weak. Temporal arteries not visible, and the carotid artery cannot be felt. Radial arteries not sclerosed. Pulse regular, good volume. Urine free from albumin. One night in May, 1911, he had intense cramp-like pain across the front of the chest. Rest and the use of digitalis were followed by distinct improvement. He reported in the spring of 1913 that he had been feeling quite well for the past two years.

Case 16.—J. C., male, aged 39 in 1908; clerk. Referred by Dr. S. Robinson.

Diagnosis.—Coronary sclerosis; syphilitic aortitis; dilatation of the heart. Lues at the age of 20. Alcoholic. For a few days prior to the time I examined him on November 10, 1908, he had been short of breath on exertion. His ankles were puffy for a short time in the fall of 1907, and again in the spring of 1908. A well-built, stout man, with grey hair, looking old for his years. Well-marked venous pulse in neck. Apex beat not seen or felt. Absolute cardiac dulness extends from right sternal border to 1 cm. beyond left nipple line. A systolic murmur replaces the first sound at the mitral area. Systolic murmur, high pitched, over tricuspid, and at aortic area loud and rough. Aortic second sound accentuated. Occasional extra-systole. Radial arteries not sclerosed.

Some œdema of legs. Urine contained a slight trace of albumin; no casts or blood. Orthodiagram showed enlargement of heart, both to right and left. Restriction of fluids and the administration of digitalis was quickly followed by a lessening of the œdema of the legs, and the urine became free of albumin. He began the carbon dioxide baths on November 18, 1908. In February, 1909, after drinking for several days, he complained of pain over the heart and great nervousness. I last saw him on April 21, 1909. He had been drinking again, and his heart was slightly dilated, and there was œdema of the ankles. He had no shortness of breath, and on April 11 played 18 holes of golf without any discomfort.

Case 17.—J. M., male, aged 62 in 1909; bookkeeper. Referred by Dr. Horace Marion.

Diagnosis.—Coronary sclerosis; auricular fibrillation; dilatation of the heart. No rheumatism. Has used alcohol and tobacco in excess. On Labour Day, 1909, while walking on the level, he had to stop frequently to catch his breath. Dyspnœa has increased. In October pulse was rapid and irregular. Œdema of the legs. For three weeks he remained in his room. For the week prior to my examination on December 10, 1909, he had been taking short walks. He was a well-built, well-nourished man. No cyanosis. Cardiac impulse increased in force. Percussion outline of heart increased chiefly to the left. No murmurs. Heart action rapid and irregular; rate 130 over the heart. Pulse of poor quality; some beats failed to reach the wrist. A tracing of the pulse showed the arrhythmia was due to auricular fibrillation. No œdema of legs. Urine contained a slight trace of albumin, but no casts. He took carbon dioxide baths in December, 1909, and January, 1910. During this time he felt quite well, and did not complain of shortness of breath. In the spring of 1910 severe cardiac insufficiency with œdema developed, and death ensued.

Action of Carbon Dioxide Baths on the Amplitude of the Pulse.—Strasburger [34], In der Stroth [32], Kommerell [31], and Swan [26] have published observations on the action of carbon dioxide baths on the pulse-pressure or the so-called amplitude of the pulse. This is obtained by

subtracting the minimum from the maximum arterial blood-pressure. The first three investigators found that in the healthy individuals they examined the carbon dioxide baths given at cool and indifferent temperatures produced, as a rule, a greater increase of the amplitude than plain water baths of the same temperature.

Swan reports four cases of cardiac disease in which the amplitude was determined in a series of baths. In one case it was increased five times and lowered eleven times; in the other four cases it was raised more frequently than it was lowered. From the data he gives I find that it was increased twenty-eight times, diminished twenty-one times, and unchanged five times. Unfortunately, he does not give any information regarding the temperature, duration, or strength in carbon dioxide of the baths. Kommerell found that the baths at or above 35° C. decreased the amplitude of the pulse, while cooler baths increased it. His observations were made on a single healthy subject.

I have collected in Table XVI observations on the amplitude of the pulse on ten patients, all of whom responded to a cool carbon dioxide bath by an elevation of the maximum blood-pressure.

TABLE XVI.

Name	Strength of bath	Temperature of bath		Duration	Before bath				After bath				Increase in amplitude
					Blood-pressure		Amplitude	Pulse-rate	Blood-pressure		Amplitude	Pulse-rate	
	Per cent.	Degrees F.	Degrees C.	Minutes	Max.	Min.			Max.	Min.			
J. D.	40	88	31·1	12	130	65	65	100	148	78	70	72	5
R. H.	100	90	32·2	10	150	85	65	80	165	88	77	82	12
W. M.	100	88	31·1	15	138	88	50	80	148	106	42	84	−8
H. R.	80	87	30·5	14	130	78	52	76	140	105	35	78	−17
D. S.	100	90	32·2	10	195	135	60	88	205	132	63	82	3
C. T.	70	89	31·7	14	130	90	40	84	140	100	40	66	0
M. G.	70	89	31·7	12	110	90	20	84	130	90	40	60	20
F. N.	100	89	31·7	10	118	75	43	83	140	95	45	82	2
D. M.	100	90	32·2	10	110	80	30	100	116	82	34	92	4
E. M.	100	89	31·7	10	118	78	40	78	122	75	47	66	7

In every instance the maximum pressure was increased by the bath, and in eight out of the ten baths the minimum pressure was raised. The amplitude of the pulse was increased in seven; decreased in two; unchanged in one. The pulse was slowed in seven; accelerated in three.

These observations indicate that a cool carbon dioxide bath usually increases the amplitude of the pulse. No special significance can be attached to this. The patient, R. H., had quite marked muscular insufficiency of the heart, and he showed a distinct increase in the amplitude. W. M., whose pulse showed a drop in amplitude, had only slight cardiac weakness. Three of the individuals (F. N., D. M., and E. M.) had normal hearts.

Conclusions.—In patients with normal hearts or with slight cardiac insufficiency the carbon dioxide baths at temperature from 95° to 85° F. usually raise the maximum arterial blood-pressure. The pulse-rate is usually lowered, but it may be increased. No direct relation between the action on the blood-pressure and on the pulse is apparent. There is considerable variation in the reaction of the circulatory apparatus in different individuals with strong hearts to the carbon dioxide baths, but occasionally a bath will raise or lower the blood-pressure one day, and the next treatment, with a bath of similar strength, temperature and duration, will give the opposite reaction in the same individual—that is, it will lower instead of raise, or raise instead of lower the blood-pressure as the case may be.

If an elevation of blood-pressure is an indication that the heart is doing more work, and a lowered blood-pressure that it is doing less work, as O. Müller maintains, then the same bath may increase the demand of the heart one day, and lessen the load another day.

The amplitude of the pulse is usually increased by a cool carbon dioxide bath, but it may be decreased. If the heart

is weak there is often a fall of the blood-pressure after a cool carbon dioxide bath, but there may be a rise. Inasmuch as some individuals with strong hearts fail to show a distinct rise of blood-pressure after a cool carbon dioxide bath, the action of the carbon dioxide bath on the blood-pressure is not a trustworthy test of the functional power of the heart.

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DISCUSSION.

Dr. MINOR: I do not like to see such interesting papers as these pass without discussion, though I should prefer not to open it. Dr. Pratt's paper interested me a great deal, since I handle a large number of patients with hypotension; most tubercular cases mean such, and because I use, not carbonic baths it is true, but cold water baths freely for their tonic effect and for their blood-pressure raising effect. Regarding Dr. Pratt's interesting experiment, it seems to me, using plain water alone, that the range of difference in temperature in his baths is too small, a difference between 90° F. for the cold and 95° F. for the warm bath being too narrow to expect any great difference in effect, if we use plain water. The effect of baths from 60° F. to 75° F. naturally is much more pronounced, and the relaxing effect of baths from 100° F. to 105° F. (I have personally tried them as hot as 110° F. on myself) is very notable. As regards the question of tension, the remarks on the use of water interested me because we all of us have a number of cases of high tension in this country due to our immoderate American living; and in these, after a short while, you are apt to have exhausted all your therapeutic resources and come up against a dead wall. You have tried the nitrites and the iodides, which latter, by the way, I still firmly believe in, and dietetic and hygienic regulation; when these fail us I believe that we will find warm water a most valuable resource. Like Dr. Daland, I have not found very much use in nitroglycerine, and I am always looking for something which will meet the needs of these cases of hypertension. They are increasing in our country every day owing to the unwisdom of the American system of life, and our high tension living, and we are getting more and more cases, and it is very essential for us to find some satisfactory and permanent means of holding down blood-pressure. There are some agencies, it is true, in which there have been reported wonderful effects, such as high frequency currents, and it is unquestionably remarkable how greatly this can temporarily reduce blood-pressure, but I have never found anything permanent in its

effect. Moreover, the patient cannot constantly be running to the electrician to be taking electric baths, but everybody has at his disposition a tub of warm water. As to whether the addition of carbonic acid in low tension cases is valuable, I do not know, but in high tension cases I am a firm believer in hot bathing.

Dr. BABCOCK: There are many points in Dr. Daland's paper which merit attention, but I shall confine my remarks to the question of the advisability of attempting to lower blood-pressure and the use of the nitrites for this purpose. I believe it is a mistake to reduce blood-pressure simply because it is abnormally high, even up to 175 or 190 mm. The criterion for treatment should be the ability of the myocardium to endure such a pressure. That is, so long as the heart is adequate our efforts should be confined to keeping the blood-pressure from becoming higher and the heart from becoming dilated and inadequate. So long as the heart is successfully enduring the abnormal pressure the individual is likely to be free from serious symptoms, and he is apt to feel weak and depressed when his blood-pressure is lowered appreciably. If, however, the heart is powerfully hypertrophied, and there is danger of rupture of a cerebral vessel, then, of course, the hypertension should be reduced, and to this end nothing is so good as blood-letting. I have ordered nitrites in many cases, and although in some by their long continued administration together with strophanthin I have seen benefit to follow, still I have never relied on the nitrites alone, nor have I been able to convince myself of their being able to produce a permanent reduction in the systolic readings. Right here I feel I should call attention to the possibility of sudden and alarming symptoms following the administration of a nitrite, owing probably to sudden dilatation of the abdominal vessels and consequent collapse. It is my conviction that, as stated by Dr. Minor, the best means of lowering blood-pressure, or at least of preventing it from becoming dangerously high, is by proper regulation of the diet together with cathartics. In all cases the articles of food containing the purin bases should be excluded so far as possible, and hence coffee, meat, and meat soups should be kept down or even forbidden. Nevertheless I recall the case of a man who, with a systolic pressure of about 190, began to manifest symptoms of cardiac distress, and who on the long continued use of sodium nitrite (1 gr. every three hours), together with half a milligram of Merck's strophanthin four times daily, at length lost all dyspnœa and other cardiac symptoms, yet without a marked lowering of his blood-pressure. In his case the diet was regulated and cathartics were used daily.

Dr. OTIS: I would like to call the attention of the gentlemen to a paper I read two years ago at Montreal in regard to blood-pressure and physical exercise. I became very much interested in the subject, and took the blood-pressure of 126 men and young women immediately before and after physical exercise, and then some twenty minutes

later. I was enabled from these observations to work out a double curve, as I called it, indicating the normal blood-pressure at the beginning of exercise, the increased maximum blood-pressure immediately at the close of exercise, and then the return to the normal or below normal a little later, depending upon the amount and severity of the exercise. These observations seemed to prove, in the first place, that the greater the training of the individual the less the variation of this curve from a straight line; and second, that if an individual were exhausted either mentally or physically and then took the exercise, instead of the rise I found that there was, in certain instances at least, a fall of the blood-pressure directly after physical exercise. This paper of Dr. Pratt's is a very interesting one, and I agree with him that you cannot draw any conclusions worth much without making several observations, and without verifying several times the same observations.

Dr. ROCHESTER: Dr. Pratt's paper was interesting to me in this respect. If we go back some years we will remember that when the Nauheim treatment of cardiac diseases was first introduced it was because of a weak heart, and that that would be restored; then there was low tension, low pressure, and things would be improved by that treatment. Then when we came to the point to where we were taking a number of observations on the blood-pressure, the Nauheim treatment was recommended to reduce the blood-pressure. It strikes me that we have in this carbon dioxide bath no use at all so far as my own observations are concerned, and reading literature, reports of cases, and the paper such as Dr. Pratt's. We can come to absolutely this conclusion, that the carbon dioxide baths have no permanent effect upon the heart at all in one way or another. The hot and cold baths, as Dr. Minor has brought out, do have an effect, sometimes a very decided and good effect, but it strikes me that this use of carbon dioxide in the water is of no use. I have found no good result from it one way or the other. In regard to the matter of the observations on blood-pressure also, of course we cannot make general conclusions from observations in regard to blood-pressure; we must consider the case. I remember as a young man, when I was very much disturbed by a symptom in a child, I called in an old physician in consultation with me. I was very much disturbed by this particular symptom. What the doctor said to me has stayed with me ever since. He said, "Look at the baby; don't look at the symptom." You have to take the whole case and observe that. Now so far as blood-pressure is concerned I have a case that has a blood-pressure regularly of 230, and she feels badly if it gets below 190. This is the case, of course, of an old lady with nephritis, but as long as her blood-pressure stays above 190 she is comfortable, but below that she is badly off indeed. We certainly should not treat the one symptom of high blood-pressure; we should always consider the case in its entirety.

Dr. Babcock: Mr. Chairman, it is very bad taste for a man to rise the second time, but I beg the indulgence of the members for a moment. I must dissent from the statement made by Dr. Rochester with reference to the effect of the so-called Nauheim baths. I have used these baths for twenty years, and I am perfectly certain that I have seen effects from the baths in the restoration of myocardial efficiency. I believe that when the baths fail to restore the more or less degree of strength of the myocardium it is because the baths have been improperly given. The lower the temperature which the patient can endure with good reaction, the more certain is one to get a beneficial effect upon the myocardium, and I am sure I use much lower temperatures than do many physicians in these baths. I have not taken blood-pressure immediately before or during or immediately after the baths. I confine my attention strictly to the examination of the heart, and I have demonstrated over and over again that when the bath is properly given and the reaction of the patient is good there is a demonstrable reduction in the area of cardiac dulness on the right side. The first effect noticed is always this reduction at the right. I do not believe in ordering baths in cases of great cardiac insufficiency, especially with œdema. I never under these circumstances use the baths until rest in bed and proper medicinal treatment has restored some degree of myocardial efficiency. Then I find the baths a most valuable agent.

Dr. Bishop: Thanking the Association for the privilege of discussing this paper, I would say that it interested me very much. As to the Nauheim treatment, I would say that I have spent two summers at Nauheim and made a very special study of the subject, and my attitude is this: I never use the Nauheim treatment except under circumstances in which it can be given with perfect technique and by one who not only understands what you might call the mathematics, but intelligently enters into the spirit of the treatment. To give Nauheim baths properly is an art—it is not a science; and if anyone has acquired the art, in his hands it does a great deal of good; but if the baths are given wrong, with bad judgment, at the wrong temperature, without discretion, and without art, they do more harm than good. They waste the patient's time, money, and everything else. I think the reason that Dr. Babcock gets results is that he gives the baths with art. Those who try to give the treatment with what scientific details can be imported from Germany are destined to failure until real skill is acquired. In New York City I know of only two or three persons whom I trust to give Nauheim treatment, and I have practically given up using it in its German form, because I find it very hard to get persons in this country to apply it properly. I do not think we can import the Nauheim treatment into this country except occasionally by the help of a few who give it properly. The underlying principles of the treatment of heart disease, as carried out at Nauheim, apart from the mere technique, are of vast

importance, and are just as applicable in one place as in another. With regard to blood-pressure, I am firmly convinced that it should be regarded only as a symptom, and that high blood-pressure often represents a compensatory phenomenon that cannot safely be interfered with as such. The injudicious administration of nitrites is more harmful than many appreciate; I believe they diminish the tone of the heart muscle, and promote the development of œdema and failure of compensation.

The PRESIDENT: I would like to hear something from Dr. Hinsdale.

Dr. HINSDALE: I agree with Dr. Bishop that you need very well balanced, experienced attendants to give these artificial Nauheim baths. If you do not, you are led into a great many errors, and bad results, of course, follow. One point which I found is this: If there is a great amplitude of temperature, if the baths are given at too low or too high a temperature, far removed from the normal heat of the body, the impression on the patient is usually unfavourable. The ranges usually adapted to cases are between 94° and 98° F. If we give as low as in the 'sixties or as high as 110° F., as has been mentioned here, we are going to get into a great deal of trouble, and I think that the action produced by a temperature of 10 degrees below normal, as, for instance, 88° as against 98° F., is far too much in the majority of cases. I think that it leaves a feeling of fatigue and taxes too much the natural power of adjustment of the circulatory system for the majority of patients; at least it is so with those with whom I have to deal. The patients that I see are usually of sensitive type, women who have had a great deal of experience at foreign spas, and sometimes at American resorts, and it is surprising to see how quickly they recognize the departure of even a single degree in the temperature of their baths. If, for instance, they are taking tub baths they instantly recognize a difference of 1 or 2 degrees from the previous bath, and remark upon it. I think little is gained by reducing the temperature of Nauheim baths more than 3 or 4 degrees below the normal standard. As the baths proceed one can reduce perhaps by a degree a day, but not to make too violent a change at first. I agree with Dr. Pratt that in most cases there is a slight rise in pressure following the use of Nauheim baths. I do not use hydrochloric acid at all, but formic acid, for the generation of the carbon dioxide. I use the 90 per cent. formic acid made in this country by large chemists, and find that it is better to handle than strong hydrochloric acid, which is dangerous for the plumbing. Whether that is so or not, I would rather use a weaker acid if necessary to generate the carbon dioxide in connection with the pound packages of sodium bicarbonate, and then, of course, use three or four or six pounds of salt in the bath of forty gallons. You are able to grade the density of the water, and at the same time, by grading the use of the carbon dioxide daily, you can increase the strength of the bath as occasion requires.

Dr. MINOR: I wish to state that I said plainly that I did not know

anything about giving Nauheim baths, but have had much experience in the use of plain water. Moreover, I should explain that in bringing the patient's baths down to 60° F., I always start at 98° F. and reduce it a degree, or two degrees, a day. Secondly, as regards baths at 110° F., I have never given these to patients, although they are used regularly in Japan, but I have taken them myself. In all these cases I make an absolute practice of bringing the water down or up gradually. At the same time I consider kidney trouble and rheumatism as common indications for the local use of cold water.

Dr. PRATT: Dr. Minor suggests that I should have used a greater variation in the temperature of the baths. My object was not the reduction or the elevation of the blood-pressure, but the study of the action of carbon dioxide baths on the blood-pressure at the temperature at which the baths are used in the treatment of heart disease. I cannot agree with Dr. Minor that the iodides reduce blood-pressure. I have used the iodides in a large number of cases of hypertonia over a long period of time, and I have not observed any decrease in the blood-pressure resulting from their administration. I do not believe a permanent reduction of the blood-pressure in chronic nephritis can be produced by any therapeutic measure, and if it could be done I am not sure that it might not be harmful as Dr. Bishop thinks. Bier pointed out many years ago that the elevation of the general blood-pressure in nephritis possibly was an aid to the functional power of the kidney. The view is generally accepted that you can lower the blood-pressure in chronic nephritis by regulating the diet. In cases uncomplicated by cardiac insufficiency I have never seen a drop of blood-pressure result from a change of diet. In heart disease the fall of blood-pressure is doubtless due to improvement in the condition of the heart, and the rest in bed and digitalis probably are the chief factors in reducing the "congestion hypertonia." Whether a restricted diet is an important factor has not been determined. In one of my cases of chronic interstitial nephritis and hypertonia with a strong heart the effect of a low protein diet was carefully studied. The food was weighed for a period of a month. There was no definite fall in the blood-pressure. Dr. Folin examined the blood in two of my cases of hypertonia without nephritis, and found that there was no abnormal increase of non-protein nitrogen in the blood. The carbon dioxide baths have been condemned in America without sufficient trial. At the present time they are held in high esteem by the leading German authorities, while in this country Dr. Babcock is one of a very small group of clinicians who are convinced from personal experience of their value in the treatment of cardiac insufficiency. This method of treatment deserves a careful trial in selected cases, and I believe it is important to observe the effect of each bath on the blood-pressure. The temperature, duration, and strength of the bath should be regulated and recorded, as all these factors influence the blood-pressure. I find that I did not make clear the test I proposed. Give the

patient a full strength carbon dioxide bath at 90° F. for ten minutes. A fall in blood-pressure is the sign of a weak heart. Normally a distinct rise in the blood-pressure results from this bath. If cool carbon dioxide baths 89° F. to 92° F. produce a fall of blood-pressure, I believe this form of treatment will do harm rather than good.

Dr. BABCOCK: When you say the temperature 90° F., will you tell me the strength of your bath as to carbon dioxide?

Dr. PRATT: The full strength bath is prepared by adding 1,000 c.c. of sodium bicarbonate and 1,000 c.c. of strong hydrochloric acid to a bath tub of ordinary size filled with water. Hydrochloric acid injures solid porcelain tubs as well as those made of iron. The substitution of formic acid for hydrochloric acid, as recommended by Dr. Hinsdale, should be tried, as I believe this modification is a distinct improvement in technique.

Dr. H. L. ELSNER (Syracuse, N.Y.): Mr. President, it will certainly prove wise and profitable to prolong this discussion because it is one which of necessity interests not only the medical profession, but the lay world. It requires brains to treat hypertension and hypotension; it requires cool and good judgment. The fact that a man presents himself with a high blood-pressure means very little, and we must brace ourselves against doing too much in these cases. It has been truly said in this discussion that the recognition of associated conditions must, of necessity, offer indications for treatment. The most rational way to reach conclusions is to classify our clinical material. In the first place there is a class of cases with high blood-pressure in which there are no associated symptoms, in which we accidentally discover the high blood-pressure. It simply means that in the absence of other symptoms and discoverable pathologic changes, we have before us a patient who demands close observation in the future. The question naturally presents itself: How shall the physician impart the knowledge which he has gained to his patient? He must do this in a way which will prevent continuous auto-suggestion, which will make it possible for him to hold the confidence of the patient that he may continue to observe the case from time to time. Most patients are upset when told that they have high blood-pressure. This has grown to be a fashionable condition, but the knowledge of its presence leads to suggestion which reacts very unfavourably. It will be possible for the physician to quietly inform his patient that he has reached the age when blood-pressure and urine demand examination at stated intervals, and he can dismiss him with the reassuring remark that "I am going to watch you." At the same time it is necessary to regulate the lives of these patients. In the second class of cases we have high pressure with sensory symptoms, sometimes angina pectoris, sometimes abdominal angina, occasionally abdominal angina and pectoral angina alternate with each other. In this class of cases the nitrites are of value. Not all of these patients have high pressure, some may even have low pressure.

The nitrites do not permanently reduce the blood-pressure; they are of value during periods of stress, for they open the periphery, relieve the heart and dilate the vessels, the seat of spasm. It is necessary for us to teach our patients with angina how to use nitro-glycerine. If they understand this thoroughly, in many cases they may prolong their lives in comfort during many years. It is not necessary to give large doses of nitro-glycerine, the small doses are efficient. I give 1/250 gr. dry on the tongue and frequently find a surprisingly prompt response in the flushing of the face and the relief of disagreeable sensory symptoms. This is by no means the result of suggestion, but there is a positive physiological action, which any man may determine for himself if he will allow an equal dose to melt on his tongue. I could report in this connection a large number of cases in which this treatment has been of benefit. These patients are instructed to take one of these pilules every five or ten minutes and rest absolutely until the spasm is relieved. Women do better than men who have threatening arterial disease, because they are less subject to the stress of daily life. Third: High blood-pressure, hypertrophy of the left ventricle with associated renal insufficiency is a complex which demands serious attention. The question arises: What are we to use in these cases? Here it becomes imperative that the life of the patient be regulated, that sensory symptoms be relieved by the nitrites and that the myocardium be given rest, and proper diet, with free use of the iodides. It must be impressed upon the profession and patient that the iodides can produce results only when given during a long period, not during a few weeks or months, but for years at a time. This question is so enormous that it is impossible to consider it fully in a single discussion or at any one meeting. There is this underlying principle which needs to be accentuated: *No case of hypertension can be regulated unless the patient is thoroughly willing to place himself under the physician's control and to act in accordance with his directions.* It is primarily a question of the regulation of the daily life of the patient. I have fallen into the habit of giving each patient in whom I find hypertension and arteriosclerosis a card upon which I write, "Don't hurry and don't over-eat." Many patients have killed themselves by over-eating. Many a man, who by the lay world and unskilled diagnostician is supposed to have died of "acute indigestion," was a victim of angina pectoris; his over-distended stomach has overtaxed his coronaries; they have proved insufficient. I wish we might prevent worry in these cases: that is impossible. The physician when he tells his patient not to worry knows how impossible it is to have that direction followed. We might as well try to stay the waters of Niagara from flowing over the rocks. We can, however, do much toward placing these patients in a healthful mental attitude. The stress and hurry of modern life, the responsibilities under which men of to-day are staggering, are the causes of degenerative processes which are robbing

us of many useful lives and are increasing enormously the number of cases of arteriosclerosis in this age. Let him who doubts this cross-index his cases, it will not require much time to reach a positive conclusion.

Dr. JUDSON DALAND: I desire to thank the members of this Association for their kindness in discussing this paper so freely, as it was for this purpose that the paper was written. I am not in accord with the view that hypertension is always compensatory. I believe it is always symptomatic and occasionally compensatory. Recognition of the cause or causes of hypertension gives plain indications as to treatment. As many cases of hypertension are due to vasomotor instability in association with neurasthenia, it is evident, as Dr. Elsner has pointed out, that the announcement to the patient that high blood-pressure exists frequently causes aggravation of symptoms; and I have often observed that worry, fret, and excessive responsibilities often lie at the root of hypertension, more particularly in those of neurotic temperament. The extreme importance of rest in these cases is well emphasized by Dr. Babcock. I heartily agree with Dr. Otis that exercise gives its maximum benefit, if the form is so selected as to interest, and give pleasure to, the patient. The benefit observed in angina pectoris by the administration of a tablet containing 1/250 of a grain of nitroglycerine is probably not due to the physiologic action of the drug, but to a quieting effect upon the mind of the patient through the taking of a drug which he believes to be a curative. As has already been pointed out, any psychical stimulation often produces well-marked influences over blood-pressure. Dr. Darlington has called our attention to the rapid increase in diseases of the cardio-vascular system, based upon a statistical study of a sufficient number of cases to warrant acceptance of his views. I believe that the presence of free carbonic acid gas in the Nauheim baths is of benefit in certain cases of cardio-vascular disease by its stimulating effect upon the skin, which is sufficient at times to cause marked redness and itching.

THE EFFECT OF CLIMATIC CHANGES ON RHEUMATISM AND NEURITIS.

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Historical.—Though there has been reference from earliest time to climate, and the influences of the same have been variously estimated, there is to-day still much confusion as to what effect meteorological changes have on certain disorders, and what particular climate is favourable and what will help some of the more common diseases with which the profession has to contend. Hippocrates speaks of how the body suffers from winds, seasons, climates and localities, and of the exposure of the Grecian cities to various winds, and of the influences and effects they have. Galen has annotated this work of Hippocrates and presented illustrations here and there. Hipparchus has written of climates, and divided them, as far as was then known, into eight, extending from Meroe on the Nile to, and including, the British Islands; he conveniently says a climate is divided between two parallels of latitude, so that the longest days of the inhabitants at one extremity exceed those of the inhabitants of the other by half an hour. The literature in reference to the effects of climate on rheumatism, gout and neuritis is astonishingly meagre, and many hours spent in reference work at the Library of Congress and at the Library

of the Surgeon-General, U.S.A., have brought to light little on the subject. Dr. Haviland, in 1855, seems to be the first to attempt to discover the influence of climate and configuration upon rheumatism.

The Weather and Rheumatism.—In the present light of the generally accepted etiology of rheumatism, this paper does not presume or attempt to show that rheumatism is caused by any other factor than an infection, but tries to draw a correlation between the primary attack and the weather and the tendency of recurring attacks to be often associated with changed conditions of the atmosphere.

Many writers have observed that cases of acute rheumatism are not distributed evenly throughout the year, and so humidity has been given much attention (Garrod, Lobert); Longstaff and Newsholme consider the relation between humidity and rheumatism to be an inverse one, and others that there is no marked coincidence between the cause of rheumatism and rainfall. It is clear there must be some reasonable basis for this discussion. Rheumatism is a disease of temperate climate, not of arctic regions, and in the temperate climates it is not always most common in winter; it is not most common at the two extremes of life—the young and the old whom you would expect climatic changes to most affect, but in adolescense and manhood when the powers of resistance are greatest; there is something more than exposure and weather, otherwise the pleura, the peritoneum, the feet and hands would mostly manifest rheumatism. In moderately low temperature accompanied by moisture are the times when we find rheumatism more prevalent. Beside moisture and temperature, ventilation seems to have much to do with the prevention of rheumatism; soldiers in South Africa, though they frequently sleep outside in damp clothes for weeks, do not suffer with arthritis. Soil and climate *per se* have little to do with rheumatism, but

may, by lessening the resistance of the individual, render the system more open to attacks of infection; too much cannot be said of ventilation and a locality that is open and airy.

Confusing the Pains of Rheumatism, Gout and Neuritis. —There is a popular idea that rheumatism, neuralgia and gout are made worse by the approaching storm.

> "Hark how the chairs and tables crack,
> Old Betty's bones are on the rack!"

In the case of Captain Catlin, which has been referred to by Dr. Weir Mitchell, the distance of the storm could often be predicted by the recurrence of neuralgia. Dr. J. M. Roddy speaks of the strange meteorological phenomena of our country in the way of magnetic storms. These, moving somewhat like a cyclonic disturbance across the continent from west to east, have also an effect upon us in some mysterious way, and patients have neuralgic and rheumatic pains intensified.

It is important that we should definitely distinguish between rheumatic fever, be it acute or sub-acute, and that large group which comprises muscular rheumatism, chronic rheumatism and neuritis. Sir William Gowers believes that often neuritis is due to affections of the fibrous tissue, such as the muscular and nerve-sheaths with their ligaments and aponeuroses, and classifies such cases as fibrositis. The typical attack of rheumatism, gout, or neuritis is very clearly pictured in the mind and the seat of the inflammation in or near the joints, in rheumatism and gout, is well understood, yet there is presented to us all at times cases of rheumatism that tell of an approaching storm, of gouty subjects whom climatic conditions affect. That climate affects the metabolism of the system is accepted; you can easier acquire an hepatic disorder in tropical climates, especially those of much humidity, than in the temperate zones; your cases of neuritis

will be more prevalent in the low lands, and still more if the atmosphere is surcharged with moisture.

The Proper Climate.—If the millennium were at hand, or we had the land of Utopia, we would order for a rheumatic a combination of dry soil, low humidity, equable temperature and an airy situation. A dry, equable climate is hard to find; the very moisture in the atmosphere which we would do without provides against frequent and wide variations of temperature; besides, the influence of a warm, moist atmosphere is at times useful in diminishing the difference between the internal temperature of the body and that of the peripheral parts. Humidity is the great factor in how we feel, for with too great dryness there will be great and sudden variations of temperature; but if the humidity is high the variations will be slight and gradual; yet few things are less inviting than a relaxing climate; one with much humidity lowers our powers of resistance by impairing our appetite and depressing our spirits and leading to lethargy.

A rheumatic should have a location of moderate altitude, where there is free exposure to sun and wind, where vegetation grows, but not in abundance, where there is dryness with moderate rainfall.

One who is merely gouty will do well in a fairly bracing climate where the metabolism is increased, but if one is definitely gouty, having heart and kidneys involved, with high blood-pressure, dyspnœa and insomnia, a climate more relaxing will be found suitable—one of a higher humidity with its consequent equability of temperature.

Most neuralgias and neuritis occur when the barometer is falling, or when it is low; the fall of the rain or the breaking of the storm generally terminates the neuralgia or neuritic attack. It has been observed that those who suddenly come out of the increased air-pressure of diving-bells or subaqueous chambers have a liability to pains in their limbs;

this at first glance seems to explain, but the translation of neuralgia cases to high altitudes does not seem to give rise to pain, nor is neuralgia as prevalent in the upper regions as on the lower levels; besides, the greater abruptness of the change from the diving-bell is out of proportion to the gradual atmospheric change.

In conclusion, the writer quotes from Dr. Weir Mitchell's article on "The Relation of Pain to Weather": "Every storm as it sweeps across the continent consists of a vast rain area, at the centre of which is a moving space of greatest barometic depression, known as the storm-centre, along which the storm moves as a bead on a string. The rain usually precedes this by 550 to 600 miles, but before and around the rain lies a belt, which may be called the neuralgic margin of the storm, and which precedes the rain by about 150 miles. This fact is very deceptive, because the sufferer may be on the far edge of the storm-basin of barometric depression, and seeing nothing of the rain, yet have pain due to the storm.

"It is somewhat interesting to figure to oneself thus: a moving area of rain girdled by a neuralgic belt 150 miles wide, within which, as it sweeps along in advance of the storm, prevail in the hurt and maimed limbs of men and in tender nerves and rheumatic joints, renewed torments called into existence by the stir and perturbation of the elements."

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NUTRITIONAL EFFECTS OF INTERMITTENT ALBUMINURIA OF ADOLESCENCE.

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In November, 1901, two brothers, aged respectively 5 and 9 years, who had been under my care since birth, developed during my absence abroad some acute infection, eruptive in character, which was termed by two competent physicians "German measles." I have suspected that they might have been cases of mild scarlatina.

During their brief illness, examination of the urine showed the presence of albumin in both instances. During their previous lives there had been no occasion for a urinary examination, hence I cannot be certain that albumin was not present earlier. Soon afterwards, however, I examined the urine of the sister of these boys, who had never suffered from any acute infection, and found on one occasion albumin present.

Subsequently, and up to the present time, I have been able to keep very careful records of specimens obtained from the boys above mentioned. For eight years both continued to present albumin from time to time, and at various hours of the day, with the utmost irregularity as to the hour.

During one month of each year, from each child I took every specimen passed in a separate bottle, noting the hour,

and succeeding in accomplishing this by having many labelled bottles in their bath-room. With few exceptions, I obtained every specimen.

By this systematic method the interesting fact developed that albumin appeared at any hour of the day, regardless of posture, diet, exercise, or prolonged rest in bed; and this albumin appeared in one specimen abundantly, while the next specimen or specimens for the earlier or later part of the day might be absolutely free.

Casts were found on one occasion only, in the older boy, and that after a hard swimming contest, which he won. Specific gravity was always within the normal range in both instances, and no other morbid elements than mentioned were ever found. Most examinations were made by special laboratory men, particularly by Dr. Nathaniel Gildersleeve, during the summer months.

In his 20th year, the older boy very seldom presented albumin, and now, in his 22nd year, it never appears.

In his 16th year, the younger showed rapid improvement, and during the past year only on two or three occasions has he had albuminuria.

These two cases seem somewhat different in their manifestations from others which I have noted in literature, and from other cases of adolescent albuminuria which have frequently come under my notice.

It is hardly correct to call them intermittent, they certainly were not orthostatic, postural, or lordotic. Many names have been applied to these irregular forms of albuminuria in the young, indicating, as literature indicates, that they have been always disturbing, confusing to the medical man, and not satisfactorily explained.

It is not my purpose to enter far into the literature of this subject, or to treat its many phases, or the chemistry of the various albumins; but rather to clarify our minds as to the nutritional effects of this anomalous state, and also to refer

to the effect of climate upon the patients whom I have had the opportunity of studying now for these many years.

It so happened that during the course of these young lives they have spent winter each year in a most balmy, subtropical climate; summers in the crisp atmosphere of Maine, upon the sea; and in the later years they have spent entire winters in the cold of Massachusetts; hence, if climate bore any relation to this form of albuminuria, a very good opportunity for discovering its effects has been presented.

The striking fact is that the amount of albumin, the extraordinary irregularity of its occurrence, one specimen loaded to an alarming extent, the very next absolutely free of serum albumin, regardless of the hour of the day, or of posture, continued uninterrupted in the moist heat of southern winters, or in the chill of the north.

Blood coagulation time was negative.

Johnson, in 1874, was the first, as far as I am aware, to call attention to the albuminuria of adolescence, naming it "cyclic."

In 1878, Dukes published observations upon eleven cases in school-boys, calling it "adolescent."

For many years after Bright albumin in the urine was considered a proof of renal degeneration under all circumstances.

In 1878, Moxon termed the condition "intermittent albuminuria," and wrote one of the earliest valuable papers on the subject, observing the entirely favourable prognosis in all cases which had not seriously damaged kidneys, which latter cases are extremely rare.

West states that albumin is always pathological, but not necessarily renal. In his opinion, the cases with serious prognosis begin after 25 years of age.

Tirard dislikes the term "functional," as he regards the condition as one of some minor, but real, disturbance in the physical economy.

Weber has never seen bad after-results in these forms of albuminuria, or record of such, and regards the lordotic as a variety of orthostatic.

Bruce had thirteen typical cases of adolescence, ten of whom were alive and perfectly well after 11 to 31 years, and had no albumin. One only died of kidney disease. He regards the condition as innocent, and considers the size of the heart, or accentuation of the second sound, as no guide in prognosis.

Goodhart refers these cases to lack of vasomotor control, which gradually adjusts itself with full physical development.

Pavy states "there is nothing to show that it is to be regarded as an early stage of Bright's disease, or that it leads to anything amiss."

Stirling examined the urine of 369 boys, and found albumin in 77 apparently perfectly healthy, mostly after assuming the erect posture; hence his term, "postural," to which Jehle had already given the name "lordotic," since he was able to produce albuminuria artificially in those children who had none by inducing an excess of spinal curvature.

Nothman increases the albumin at will in actual renal disease by artificial lordosis.

Schreiber and Menge have found albumin for some hours after palpating a movable kidney, otherwise normal; and Sutherland notes the frequent coincidence of albumin and movable kidney in children, and remarks that "this variety of albuminuria may be described as cyclic in character, postural in origin, and adolescent as to the period of life."

Wood has seen various cases in which the lower lobe of the right kidney was easily palpable on inspiration, and in all intermittent albuminuria was present.

Dukes, physician at Rugby, gets albumin in 16 per cent. of all boys entering the school between 13 and 14 years of age, and Fetherston obtained albumin in 42 out of 350 boys examined.

Ullman, in his Berlin studies, found albuminuria in 33 per cent. of adolescents; and Langsten in 5 to 12 per cent. of the young, cyclic in character.

Langsten and Baginsky both find cyclic, transitory albuminuria common after various infectious diseases.

Holt states that many youths with transitory albuminuria are well nourished and have no sign of disease.

Goodhart and Still, 1910, "know of no evidence that renal disease results."

Koplik remarks that 80 per cent. of these cases occur before the 20th year, and regards it a condition "of metabolic activity and growth," and its significance induces wide diversity of opinion.

Rachford, in 1912, says it is "comparatively unimportant" and "has little or no pathological significance, as the findings are very common in normal children."

Ellis found 5·4 per cent. albumin in a man, aged 26, who had eaten excessively of eggs. The albumin permanently disappeared on discontinuing this form of diet.

Osler states that the presence of albumin in any form under any circumstances is indicative of change in renal or glomerular epithelium, which may be transient, slight, and unimportant, dependent upon changes in circulation, &c., as in the so-called functional cases. He states that a large proportion of these cases get well.

In the instance of the two brothers, to whom I have especially referred, I have been unable to observe any nutritional fault, other than occurs in the ordinarily healthy child; the older has passed through fitting school and college successfully, with high rank. He has been subject to occasional colds, like other children, and once had a sharp attack of influenza, which continued with fever for a fortnight. Physical signs at that time showed a small pneumonic patch at the second right interspace, which entirely disappeared. Otherwise there has been nothing to

note. For physical strength and activity of body and mind, for complete growth, both in height and weight, both boys are above the average. The younger brother, on one occasion, had furunculosis persisting for some months, cured by autogenous vaccine. At the height of his fitting school work, in the most difficult year, he expressed himself as languid and weary, and required more than the usual amount of sleep; but he did not lose flesh, and as this disturbance in his sensations was not accompanied by loss of appetite or weight, and left no impression after the immediate work was finished, it appeared to have no significance other than similar conditions would have had in a perfectly sound child.

From the opinions of the authors above mentioned, as well as from my personal experience, not only with the two cases detailed, but in many others which I have been able to follow for a lesser period of time, one is led to the conclusion that transitory albuminuria, intermittent or remittent in character, has little or no effect upon the nutrition of the child. We meet with many children, who suffer from varied forms of malnutrition, in whom albuminuria is never present; in proportion a much larger number indicate malnutrition without albumin than with it.

It must, of course, be accepted that transitory albuminuria continuing for years, though in adolescents, whether primary or succeeding some mild exanthem, requires watchful care, and in no individual case should one assume that the ultimate result may not be permanent injury to renal structure, but it is significant of the comparative, or complete harmlessness, of the condition discussed, that as one follows literature from the earlier imperfect studies, to the latest complete analyses, opinion has crystallized in the minds of authors and students of children that we are dealing in most instances with a harmless and passing phase of adolescent life.

WHAT RELATION, IF ANY, HAVE THE FAUCIAL TONSILS TO PULMONARY TUBERCULOSIS?

By E. FLETCHER INGALS, M.D.
CHICAGO.

In 1878, Cohnheim suggested that tuberculosis of the cervical glands might be the result of primary tuberculosis in the buccal or pharyngeal mucosa or tonsils. In 1879 Orth, in some feeding experiments with guinea-pigs, produced tuberculosis in the cervical glands, and in one in the tonsils. Baumgarten confirmed these results in 1884. Cornil and Ranvier the same year described the lesions in tubercular tonsils in their manual of pathological histology; and Strassmann the same year mentions the frequency of tonsillar tuberculosis in phthisical patients (thirteen in twenty-one cases). This seems to have been the beginning of the special literature on this subject. In 1885 Abraham wrote on tubercle of the tonsil, and in 1891 Dmochowski concluded that tubercular deposits in the faucial and lingual tonsils often resulted from auto-infection. In 1892 Conoreur described the probable relation of tuberculosis of the tonsils to the same disease in the cervical glands. Schlenker, in 1893, published observations on man, which appeared to indicate that the tonsils are sometimes infected primarily by bacilli in food, but more often by bacilli in sputum from the lungs. Krückmann, in 1894, confirmed the observations of

Strassmann, Dmochowski and Schlenker, and Dieulafog, in 1895, showed that tuberculosis of the tonsils often existed without cervical symptoms. For this early history of this subject I am indebted to Jonathan Wright, who kindly furnished me advance copy of his second edition of "A History of Laryngology," from which this has been compiled.

An article calling attention to the possibility of tubercular infection through the tonsils, by Professor G. Sims Woodhead, appeared in the *Lancet*, October 27, 1894. He endeavoured to show that the faucial and pharyngeal tonsils are important channels of infection in this disease. He showed also that the lymphatic glands of the neck may become infected with tuberculosis through the faucial tonsils while the tonsils themselves may not be involved in the process; that is, the tubercle bacilli may find entrance through the tonsil and pass on by the lymphatics to the glands while the tonsils may escape injury. Subsequent observations seem to have confirmed this view, although Cheyne (*Brit. Med. Journ.*, 1899) contended that the faucial and pharyngeal tonsils did not play an important part in the ætiology of cervical adenitis.

Jonathan Wright (*New York Med. Journ.*, 1900) pointed out the liability to error in the various methods for detection of tuberculosis in lymphoid tissue; for it is very difficult to detect tubercle bacilli in these structures even when they are studded with tubercles visible to the naked eye; and it is the exception to find them in the scanty anatomical tubercles of latent tuberculosis.

Arthur Latham, the same year (*Lancet*, December 22, 1900), states his belief that the primary infection in tuberculosis is by the lymphatic system, and that it spreads from the cervical to the bronchial glands. This view, however, appears to have been refuted by subsequent research. In a

personal letter of November, 1912, Jonathan Wright says: "I think it has been pretty conclusively shown that the infection is not through the cervical lymphatics, which do not communicate with the pulmonary lymphatics," but that the whole of the infection "is poured into the general circulation."

Nevertheless, Latham's views of the ætiology of pulmonary tuberculosis are in large part correct according to most pathologists of the present time. I quite agree with him in the statement that "There can be no doubt that predisposition, hereditary and acquired character of the soil are most important factors."

In the letter already referred to, Jonathan Wright says in connection with the quotation given above: "This makes the infection of the lungs, so far as the agents of it go through the tonsil, an affair of local predisposition."

Latham states that tuberculosis of the bronchial glands is the most constant lesion found, and that in the majority of cases the tubercular process is more advanced there than elsewhere. But he thought the lungs were most frequently involved first, and that the disease spread by the lymphatics to the bronchial glands. However, he believed that the primary infection was by the lymphatic system. He thought that when the bronchial glands became involved first the disease might spread from them to the lungs by continuity, by ulceration into a bronchus or into a blood-vessel, or by the lymphatics, but against the supposed lymphatic stream. He states that in children under $3\frac{1}{2}$ years of age pulmonary tuberculosis is usually dependent upon infection of the bronchial glands, from which it spreads to the lungs; but that after four years of age, as in adults, the lungs are involved first, and the process spreads from them to the bronchial glands.

In the *Practitioner,* 1901, StClair Thomson calls attention to the infrequency of involvement of the nares in patients

suffering from pulmonary tuberculosis (only once in 450 cases), although it is generally conceded that the infection is usually aerogenous.

In autopsies of 100 cases of pulmonary tuberculosis, the pharyngeal tonsil showed tuberculosis in only 6 per cent. on microscopic examination, although inoculation experiments demonstrated its presence in nearly 20 per cent. The larynx was involved in 30 per cent. The discrepancy between different observers is due largely to the methods of examination. As already shown by Wright, tubercle bacilli are difficult to demonstrate in lymphoid structures, although the histological changes may be very apparent. In the examination of seventy-eight cases of pulmonary tuberculosis, Brieger found no tubercle bacilli either on the surface of the tonsils or in their crypts, but histological evidence of the disease in the tonsils was present in 6 per cent. Thomson believed that the infection could take place through the tonsil, but did not think this common, and he called attention to the experimental results on pigs by Sidney Martin, which demonstrated that the infection could take place through the tonsil without involving this organ in the tubercular process.

J. L. Goodale (*Brit. Med. Journ.*, 1903) claims that chronic absorption of bacterial products of decomposition is not likely to occur through the pharyngeal or lingual tonsils, but thinks it does frequently take place through the faucial tonsils.

L. Kingsford (*Lancet*, 1904) states that tuberculosis of the tonsils is comparatively frequent, but he does not think this often the primary seat of the disease. He considers tuberculosis of the tonsils usually secondary to foci in other places, and thinks the tonsils, especially in adults, become infected through the sputum or by the blood-stream. However, he states that primary infection of the tonsils is not uncommon in children, in whom he thinks it might occur either by inhalation or from infected food. In the same

connection it is stated that in children Friedman found evidence of tuberculosis in 11 per cent. out of 145 cases, and Latham, by inoculation experiments, found it in 15 per cent. of 48 cases; while Schlenker and Wright had found tubercle bacilli in adjacent cervical lymphatics, which seemed to have passed through the tonsils without any involvement of these glands.

Geo. B. Wood (*Journ. Amer. Med. Assoc.*, May 6, 1905) states that secondary infection of the tonsils takes place readily in pulmonary tuberculosis. Seven different observers, in examining 136 bodies dead of pulmonary tuberculosis, found the tonsils also involved in 69 per cent. However, twenty-three different observers, in examining 1,671 cases, found primary tuberculosis of the tonsils in only 5·2 per cent. He believes that in the great majority of cases of *pulmonary* tuberculosis, few, if any, are caused by infection through the tonsils. He believes that in 90 per cent. of the cases of tubercular cervical adenitis in children the faucial tonsils were the portals of entry. He states that it seems scarcely possible that tubercle bacilli could gain entrance through the normal mucosa of the mouth and fauces, which is covered with squamous epithelium especially adapted as a protective covering. The surface of the tonsils is covered with the same epithelium, but in the invaginations which form the crypts the epithelium is so changed that it loses this normal protective function.

If Wood is correct, there could be very little if any entrance of infective material through the mucous membrane covering the normal tonsil in which the crypts are negligible; but with enlargement of the tonsil and deepening of the crypts the opportunity for the entrance of bacilli would be very greatly increased. In other words, absorption of tubercle bacilli is not at all likely to occur through the tonsils, excepting when they are enlarged. The larger the gland the

deeper the crypts, and there must be a correspondingly increased chance for absorption of any infective organisms or other minute substances.

J. L. Goodale (*Boston Med. and Surg. Journ.*, Nov., 1906) calls attention to the demonstration by Theobald Smith that in cattle tubercle bacilli may enter the system through the mucous membrane of the mouth and throat without leaving any visible trace at the point of entrance. He also points out the difference in behaviour of cervical adenitis of tubercular and non-tubercular origin under certain conditions. He states that tubercular cervical adenitis may exist in association with tubercle bacilli in the tonsils with or without visible changes in these organs; and that removal of such tonsils may have no effect on the cervical glands. But there are other cases of cervical adenitis associated with distinct enlargement of the tonsils in which the irritating material seems to be generated in the tonsils. In these cases removal of the tonsil is followed by prompt relief. From the foregoing it would appear that removal of enlarged tonsils may tend to prevent the absorption of tubercle bacilli, but that after the damage has been done this operation may have no influence on the tubercular cervical glands.

In the *Transactions of the American Laryngological Association*, 1907, J. L. Goodale states that the faucial tonsils are the most vulnerable point of entrance for micro-organisms, and that previous pathological alterations in these structures favour such infections. Yet he thought that disproportionate emphasis had been laid upon these pathological alterations in comparison with the receptivity of the host. I quite agree with him in this view, and fully believe that receptivity or resistance of the host is by far the most important fact in the development of tuberculosis. The tubercle bacillus seems constantly with us, and it has been shown to be present in 97 per cent. of practically all adult

human beings. It probably is present in every one, whether or not there have been pathological changes in the mucous membranes. But if the individual's resistance is good it has little or no effect; whereas, whatever lessens this resistance, be it diseased tonsils or anything else, favours the development of more or less serious tubercular processes.

Jobson Horne (*Journ. of Laryng., Rhin. and Otol.*, 1907), considering the channels of tubercular infection, asserts that the infection may occur through the tonsils from food; but he thinks that in the vast majority of cases infection of the lungs takes place through tubercle bacilli in the inspired air, either directly or secondarily through the bronchial glands.

Osler ("Modern Medicine," 1907) says much has been written of late about the relation of the tonsils to tuberculosis, especially with regard to its importance as a portal of entry, but even yet, after the study of hundreds of cases, there is no great unanimity of opinion. He quotes figures which show primary infection of the tonsils in from 3 per cent. to 6 per cent. of 200 cases studied by Piffe and Rethi.

In the *Johns Hopkins Hospital Bulletin*, November, 1908, Goodale is quoted as having found quite a large percentage of tubercular tonsils associated with cervical adenitis, which was demonstrated by cleaning up of the glandular condition on removal of the tonsils. This does not appear to have been the common experience, although very many physicians, since Heberden's observation on rheumatism in 1804, have demonstrated the relation of the tonsils as a portal of entry to the infection in various diseases.

In the *British Medical Journal*, November 19, 1910, p. 1622, the relation of enlarged tonsils to tuberculosis is discussed, but nothing new is added.

E. C. Sewall (*Journ. Amer. Med. Assoc.*, September 9, 1911, p. 868) states that between 1,600 and 2,000 cases of

primary tuberculosis of the tonsils have been studied by various observers which have accounted for a little over 5 per cent. of all cases of tuberculosis coming to autopsy. He states that, excepting when ulceration has occurred, there are no distinguishing clinical signs of tuberculosis in the tonsils.

In 772 pairs of enlarged tonsils removed and examined in the laboratory of the Cooper Medical College, only 3·9 per cent. were found to be tuberculous. As Naegeli found tuberculosis in 97 per cent. of all adult bodies, this small percentage in diseased tonsils appears to have no significance whatever, and the only wonder is that a much larger percentage was not found.

As showing the relation of infected tonsils to cervical adenitis, he states that of 160 cases of diseased tonsils the cervical glands were also slightly or much affected in 68, and that in three of these both the cervical glands and the tonsils were found to be tubercular. In 57 of these the enlarged cervical glands disappeared when the tonsils were removed. In six they subsided, but returned; but in five the cervical adenitis was not benefited by the operation.

In the *Journal of the Indiana State Medical Association*, November 15, 1912, p. 467, it is stated that it has been repeatedly proven that in a hundred children having enlarged tonsils and adenoids, there are appreciatively fewer cases of tuberculosis than in a hundred children with normal throats. The significance of this, if true, is obvious, but I have been unable to verify the statement, and the author of the article has failed to respond to my personal letter of inquiry, therefore I am led to believe there has been some mistake.

I have in my files of histories of private patients, over 25,000 carefully kept records, more than 10 per cent. of which are of tuberculosis. In these cases there were made careful examinations of the nose and throat, as well as of the chest, and the histories were carefully taken, for heredity, previous

diseases, enlarged glands, general condition at the time of the examination, and indeed everything that might have any bearing on the origin or course of the disease. Dr. W. S. Bracken, one of my associates, employing all his spare time for several months, and assisted by some of the other physicians in my office, has gone over these records carefully, and analysed the findings with a view to discover, if possible, a relation between the tonsils and laryngeal or pulmonary tuberculosis.

He took at random 100 cases (with no selection excepting to assure himself that the blanks had been thoroughly filled out), thus giving a fair average of the whole. From the records of non-tubercular patients who consulted me for various diseases having no relation to tuberculosis, such as functional and organic disease of the heart, asthma, simple bronchitis, pleurodynia, emphysema, pharyngitis, rhinitis and laryngitis, he took at random 100 other records for control, thus making a fair analysis of my case records of about 25,000 different private patients. These records are far and away more complete and accurate than those of hospital patients. His analysis shows that 26 per cent. of the tubercular cases had some macroscopic change in the tonsils, varying from slight enlargement to marked hypertrophy, ulceration, or other evidence of disease. In only 4 per cent. was there any record of cervical adenitis, and in only one of these was there any change in the tonsils, which were noted in that case to be slightly enlarged.

Among the control cases 46 per cent. had enlargement or other evidence of disease of the tonsils, nearly twice as many as in tubercular cases, and not one of them had cervical adenitis. So far as these records go they show that affections of the tonsils are much more frequent among non-tubercular than among tubercular patients; but that cervical adenitis, while comparatively infrequent (4 per cent. only) in tubercular

patients, is much less common in non-tubercular cases. It is a matter of surprise to me to find that in only one of the cases of cervical adenitis was there even the slightest evidence of disease of the tonsils.

Ten or fifteen years ago it was quite commonly believed that disease of the tonsils was a frequent cause of pulmonary tuberculosis; but subsequent research appears to have proven that tubercle bacilli may enter and pass through the tonsils and cause disease of the cervical glands, while the tonsils themselves may escape all injury; and this research has also shown that there is no direct connection between the cervical glands and the pulmonary lymphatics, and therefore, that involvement of the lungs associated with cervical adenitis must be a systemic infection rather than a result of the glandular disease.

From a thorough study of the literature, and from an examination of my own records, I am forced to accept as correct the consensus of opinion which now fully sustains the personal views of Jonathan Wright, who wrote me as follows in November, 1912: "To tell the truth, I do not believe there is any relation of the tonsils to pulmonary tuberculosis."

WHEN IS A TUBERCULOSIS PATIENT CURED?

By LAWRASON BROWN, M.D.
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Having been closely associated with sanatorium work for a number of years, I have long been interested in the rather academic question of classification of the results of treatment in pulmonary tuberculosis.

The historical side of the question dates as far back as Hippocrates (about 400 B.C), who is quoted as believing in its curability. From that time to this the subject has been debated by many authorities, who opposed and upheld the curability of the disease.

"The cases contained in Bayle's works," Laennec writes, "as well as what I have myself stated above concerning the development of tubercles, prove sufficiently that the idea of the possibility of curing phthisis in the first stage is an illusion. Crude tubercles tend essentially to increase and soften. It is perhaps in the power of art to check their development, to suspend their rapid course, but not to compel it to retrograde. But though it is impossible to cure phthisis in the first stage, a considerable number of facts have proved to me that, in some cases, a patient may recover after having had in his lungs tubercles which have softened and have formed an ulcerous cavity.

"Finally, recovery in cases of pulmonary phthisis," he

continues, "where the organ has not been wholly invaded, seems to me to present no character of impossibility, either as regards the nature of the disease or that of the organ affected; since tubercles in the lung differ in nothing from those which, being situated in the glands, take the name of scrofula, and the softening of which, it is well known, is very often followed by a perfect cure."

At no time has the discussion waxed so warm as since the widespread erection of sanatoria which have published yearly reports. At first the public had to be persuaded and convinced that pulmonary tuberculosis was curable, or it was said that they would not contribute to the support of such institutions. The use of the term "cure," when applied to many patients who later relapsed and succumbed to the disease, have caused many men who have followed a large number of patients for a period of ten years to doubt the wisdom of the use of the term "cured" as applied to pulmonary tuberculosis.

Permit me at this point to call your attention to the word "cure." "To cure" is defined as "to restore to health or to a sound state." This means, I take it, when applied to a disease, a restoration of the body to a state in which no vestige whatsoever of that disease exists within the body, although in certain instances some scarring is left behind.

Now in many instances disease runs a limited course, and results in either death or cure, and that in a matter of days or possibly of weeks. To this class belong many of the infectious diseases of childhood, small-pox, pneumonia, cerebrospinal fever, whooping cough, cholera, tetanus, &c. When the patient is restored to full vigour he is, according to the foregoing definition, "cured" of his disease, as none of the infectious organisms can be demonstrated to be harboured in his body.

There is, however, another class in which some diseases

may be placed where it is exceedingly difficult, even by the most refined tests, to determine positively whether or not the infecting organism has been completely eradicated from the body. Here we may place malaria, gonorrhœa, syphilis and tuberculosis. The gynæcologists bear witness to the obstinacy of gonorrhœa in the male, and its awful results in many innocent women. The discovery of the *Spirochæta pallida* in the cortex of the brain in paresis is but one instance of the folly of pronouncing a man "cured" of a disease which long lies dormant.

I have stated it to be my belief that, when clinical evidences of disease are once manifested and the specific organisms demonstrated to be present in a patient, he cannot be said to be cured until it can be shown that these organisms no longer exist in his body.

It must be clearly understood that reference is made in this paper only to patients in whom pulmonary tuberculosis has been clinically manifested. Those individuals who suffer only from tuberculous infection need not be considered at this time, though none can gainsay that certain conditions may not at any time so reduce the resistance of these individuals that the latent infection may break out as an acute clinical disease.

Many of us to-day think in terms of pathological anatomy, and when "cure" of a pulmonary disease is mentioned, pneumonia as a type occurs to most of us. Here grave pathological alteration takes place, but apparent perfect anatomical recovery of the organ is the rule. The pathology of pulmonary tuberculosis is far different. Even when a tubercle is only just visible to the naked eye, retrogression without scar formation is exceedingly rare, if indeed it ever occurs. But even so, the work of Hodenpyl has shown that cure can occur and only a scar remain. Consider again a larger, possibly a confluent tubercle, which has advanced to

caseation. Two reparative processes are now possible; first, replacement of the caseous matter by fibrous tissue; second, calcification of the caseous mass. It would seem that either process should lead to cure. Fibrosis may do so, but only when it completely replaces all caseous matter, which may contain tubercle bacilli as long as it exists. This occurs more rarely than some believe. Further, the presence of minute, fresh tubercles about such encapsulated masses has often been observed. Calcification of a tubercle is no proof that it does not contain tubercle bacilli, though in man tubercle bacilli occur less frequently than in other animals.

That a well-defined cavity can ever fully heal without leaving at least a fistulous cicatrix is highly improbable, but it is quite possible that in such patients, with long "ultra-chronic" disease, the tubercle bacilli may finally disappear. Such patients, if they do recover, are much like the man with an amputated limb.

Viewed, then, from the pathological standpoint, the longer the disease persists the less is the chance of cure, and the further the disease advances the more unlikely the possibility of cure becomes, which may be looked upon as a corollary of the first statement.

So far no proof has been adduced to show that one attack of pulmonary tuberculosis leaves behind it any immunity to the disease. Susceptibility to the tuberculous poison of a person once infected with the tubercle bacillus may persist for a long period. This may last after all the tubercle bacilli in the body are destroyed. If such occurs, it is readily seen that susceptibility to tuberculin must not necessarily be lost with the disappearance of tubercle bacilli, and, on the other hand, the lack of reaction to tuberculin is no proof, as is well known, that tubercle bacilli do not occur in the body. So far as I am aware, no antigen has as yet been obtained that reacts often enough with the serum of the

tuberculous, to render diagnosis by the method of complement deviation of any practical value, or to detect the absence of tubercle bacilli in the body.

The variability of physical signs in pulmonary tuberculosis is notorious, and to attempt to draw "pathological" conclusions from them is totally unwarranted. The fluoroscope or radiograph will, in most instances, reveal far more extensive disease than the physical signs indicate. Loss of physical signs, then, may mean only that the lesion is deep-seated. In few cases, however, have I been able to record a complete disappearance of physical signs, and we must consider what their persistence indicates. When the pulmonic changes on examination are slight, it is possible that scarring in the lung may produce them, but even this arouses doubt, and I believe that the signs of a "healed" lesion are practically better interpreted as those of an "arrested" lesion.

The relation of symptoms to the state of the pulmonary disease or physical signs is also notoriously uncertain. One patient may have extensive physical signs and no symptoms, and another no physical signs and severe symptoms. Since this is the case, the mere absence of symptoms indicates neither loss of physical signs, disappearance of foci of disease, nor, most improbable of all, absence of tubercle bacilli in the body. Finally, the disappearance of all symptoms and physical signs may occur, and yet the radiograph show well-defined foci in the lungs.

These facts have all been clearly recognized, and the time element has been introduced into all systems of classification. Some would affirm that loss of all symptoms, while at work under ordinary conditions of life for a period of two years, is sufficient proof that the disease is cured. However, many of these patients have persistent physical signs, and later fall ill again of pulmonary tuberculosis. Such

patients surely are not cured. From this it seems clear to me that we are not justified in basing a conclusion that the patient is "cured" upon the loss of either physical signs or symptoms or both. I have long held that only at autopsy can we pronounce a man cured of clinical pulmonary tuberculosis. That his disease is arrested lapse of time has shown, but at what point he passes from a state of arrest to that of cure I believe no man can say, though I believe such patients can be cured.

Many words have been prefixed to "cured," showing one and all that the foundation of belief in the term rested not upon solid rock. We hear of "apparent" cures, "relative," "economic," "practical," "working" cures, and so on. Those who use these terms confess their doubts and may be passed by. On the other hand are those who for "the good of the cause" (the eradication of tuberculosis) still believe that the general public needs pap and not strong meat, honied words, and not real truth. They advance expediency to uphold their position and fear the loss of funds for sanatoria. To these I can only point to the work of a man who, through long experience, saw the light when many of us were still in darkness. I refer to Dr. V. Y. Bowditch, who for years never used the term "cure," and now uses it only under protest. Yet I have never heard it intimated that Sharon had any more difficulty in raising funds than any other similarly situated institution. Those who argue expediency build, I am convinced, upon the sand.

I have not mentioned the death-rate of the "apparently cured," which at the Adirondack Cottage Sanitarium is three times that of the general population. I do not believe, though I have no figures to prove it, that the death-rate of the "cured" cases is much lower. The results in dispensary work, I imagine, are not very different.

So far I have not considered the question from the side of the patient. That there are two ways of looking at this I am well aware. So to discourage a patient that he will not fight is serious, but to give him false confidence by the use of terms he misinterprets is still more grave. I believe in being as frank as the intelligence of the patient permits, but the same truth may be told in two ways. I doubt if more than one or two, if indeed so many, of my patients have lost any ground through what I have told them, and I feel sure that many have been saved from serious blunders which might have been brought about by considering that "apparently cured" in pulmonary tuberculosis was equivalent to "cured" in measles.

In conclusion, as a confession of faith, I may say that I believe that pulmonary tuberculosis is curable; that spontaneous healing of tuberculosis infection occurs frequently; that healing of clinical tuberculosis may, but rarely does, occur; that permanent arrest is the most frequent termination of the disease in those who are later able to follow their usual occupations; that temporary arrest, of longer or shorter duration, occurs in all except the acute cases, and that continuously progressive disease follows only upon several temporary arrests.

CASES OF TUMOUR OF THE LUNGS AND MEDIASTINUM SIMULATING PULMONARY TUBERCULOSIS.

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Several cases of intrathoracic tumour, which had been mistaken for pulmonary tuberculosis and sent to Colorado on that account, have come under the observation of the writer.

It is by no means desired to criticize those who made these diagnoses, for it is well known that, early in their course, these cases may simulate tuberculosis so closely and the physical findings be so similar or indefinite as, in some cases, to make differentiation extremely difficult or even impossible.

Case 1.—*Lympho-sarcoma of the Mediastinum.*—Mrs. H. M. G., aged 30, seen the day of her arrival in Colorado. Family history not important. Well till past year, when she felt run down and nervous; cough, expectoration, sweats, fever and chills. Much emaciation. Glands in neck and axillæ swollen and tender. Vague pain in front of right lower chest early in illness; none since. No dyspnœa. Greatly troubled by itching over whole body. Had had local treatment for supposed tubercular laryngitis.

Arrived in Colorado desperately ill, with family of five, having sold their home to realize sufficient funds for the journey.

Physical examination showed enlarged glands above right

clavicle; flatness in right front, from fifth rib to near iliac spine and to umbilicus. Abdomen tense and very tender over this area. Also area of dulness corresponding to lower half of sternum, continuous with cardiac dulness, and extending half-way to right nipple. Absence of respiratory and voice sounds over this area.

Right back showed signs of fluid up to lower fourth of scapula. No other abnormal signs in the chest. Dilated, deep epigastric veins on either side. Sputum on one examination showed no tubercle bacilli. Died uræmic after ten days.

Autopsy showed a tumour the size of a child's head in lower anterior mediastinum, with metastatic areas in both lungs and considerable sero-fibrinous exudate in right pleural cavity. No macroscopic evidence of tuberculosis anywhere.

The liver was greatly enlarged and pushed down to the brim of the pelvis. Spleen somewhat enlarged.

Microscopic examination of the tumour showed it to be lympho-sarcoma.

Case 2.—Primary Carcinoma of Lung.—Mr. J. H. B., aged 53, merchant. Came to Colorado in December, 1908. Family history not important. Previous history good, except for hæmorrhoids and stone in the bladder.

Cough with slight expectoration for four months. Lost 28 lb.; moderate fever. Sputum had been blood-stained a few times, but had showed no tubercle bacilli on several examinations; positive skin tuberculin test. Chest examination had caused only a suspicion of trouble in left lung.

Physical Examination.—Looks very pale and ill. Chest shows slight dulness in left front from second to fourth rib, and from left border of sternum to nipple line; fine moist râles after cough, with harsh respiration over this area; no change in voice.

In left back harsh respiration over whole inter-scapula space, and slight dulness over lower half of area noted. No other abnormal signs.

X-ray Examination.—Right side: apex clear, enlarged bronchial glands; heart somewhat to the right.

Left side: apex clear, few nodules from clavicle to second rib; from second rib to bottom of the fifth is a distinctly outlined consolidated area, which is being excavated. Several examinations of sputum failed to show tubercle bacilli. At

the end of a month the signs in chest had become more marked; the patient became restless, and spent two months travelling in Arizona and California. Returned March 15, 1909, having lost much ground in every way. Nearly the whole left chest was flat on percussion, with respiration diminished above, bronchial in type, and absent in lower portion; heart displaced to right. Aspiration of 30 oz. slightly turbid fluid, not blood-stained, and showing no tubercle bacilli or other organisms, but a definite predominance of lymphocytes.

The aspiration gave little relief from dyspnœa. From this time till his death, April 16, 1909, he had rapid respiration, gradually increasing dyspnœa, moderate fever, slight expectoration which was free from blood and tubercle bacilli on numerous examinations, and marked weakness. Each of four aspirations yielded about 30 oz. fluid of the same character as described above.

Autopsy.—Left lung was simply a tumour mass, in areas softened, but mostly hard; pericardium, upper portion of the pleura, and of the lung were practically inseparable and indistinguishable, and firmly adherent to the chest wall. The mediastinum had a considerable mass of enlarged glands. The right lung showed no evidences of tuberculosis, but an area of metastasis near the apex, with hypostatic pneumonia at the base.

There were metastases in the liver and spleen, found only on section.

There was a chain of mesenteric and post-peritoneal glands extending from the bifurcation of the aorta to the diaphragm, but no other lesions in the abdomen. Unfortunately, the specimens were lost and there was no microscopic examination, but it appears to have been a case of primary carcinoma of the lung.

Case 3.—*Persistent Thymus; possible Hodgkin's Disease.*—Mrs. C. P., aged 40. Family history not important. Never strong; much pelvic and digestive trouble for years; always sallow and anæmic; suspicion of gallstones several years ago. Eleven years ago spent a winter in Florida because of cough and weakness. Two years ago, during severe illness of a son, lost much weight, and never well since. Eighteen and six months ago had pain in chest, which required strapping.

For a year cough with slight expectoration, fever, and a few night sweats. One year ago glands removed from right side of neck. No microscopic examination, but considered by a competent surgeon to have been tubercular. Said to have had moist râles at both apices, but sputum not examined. Menses scanty.

In bed past two months, with afternoon temperature 99° to 101° F.

On arrival felt very weak, considerable dry cough, moderate dyspnœa, and much indefinite abdominal discomfort.

Physical Examination.—Poorly nourished, very pale and sallow, but scleræ white; skin almost bronzed throughout, but not especially in axillæ or groins, and not seen on mucous membranes. Says this discoloration has been present many years, though some pigmented spots on abdomen and loins are recent.

Systolic pressure 112. No enlarged glands except one above right clavicle, about the size of a pigeon's egg, movable, firm, and not tender.

Spleen and liver considerably enlarged, and the latter is very tender. Indefinite tenderness in right lower quadrant. Uterus large, retroverted and tender. Hæmoglobin, 65 per cent.; reds, 3,400,000; whites, 12,500; colour index, 1. Stained film, negative. Urine showed a trace of albumin and a few hyaline casts. Wassermann test, negative. Heart, negative; lungs, negative, except moderately diminished respiratory murmur at right base behind, and slightly harsh respiration in right front, in the third interspace.

For a time the patient seemed to improve with large doses of quinine and cacodylate of soda, but on the whole she gradually grew weaker and lost much flesh. The temperature varied from 99·5° to 101° F., and pulse was rapid. Respiration was not rapid nor embarrassed, and there was but little cough till shortly before death, when there was distressing paroxysmal cough with attacks of dyspnœa. There had also appeared dulness and a systolic murmur over the upper portion of the sternum.

X-ray Examination showed a shadow in upper mediastinum, centrally located, $7\frac{1}{2}$ cm. wide at second rib, front, and extending to the upper border of the clavicles. The heart shadow was normal in size, shape and position. There

was no evidence of tuberculosis or other involvement of the lungs.

The fluoroscope seemed to show expansile pulsation of the mediastinal shadow.

Autopsy.—A hurried autopsy, after the body had been embalmed, showed in the upper mediastinum a mass about the size of the first, composed partly of a persistent thymus, $2\frac{1}{2} \times 1\frac{1}{2}$ in. in size, and partly of firm, discrete lymph-glands, which surrounded it and extended about the roots of the lungs. The individual glands varied in size from a cherry to an English walnut. The trachea and œsophagus, though somewhat surrounded by the mass, were not involved.

Heart and aorta normal. Pleural cavities smooth. Lungs showed no macroscopic changes.

The peritoneal glands formed a mass about twice the size of the pancreas, the individual glands being discrete, and measured up to $1\frac{1}{2}$ in. in diameter.

Both liver and spleen were enlarged, the cut surfaces showing nothing but the usual evidences of passive congestion.

Stomach, intestines, pancreas, and kidneys negative.

Microscopically, the thymus showed small scattered lymphoid areas surrounded by fatty tissue, and areas of calcareous deposit.

The lymph-glands, on numerous sections, showed nothing but a diffuse hyperplasia.

Case 4.—*Malignant Disease of Lungs.*—Male, aged about 50, seen in consultation shortly before his death. Had been supposed to have pulmonary tuberculosis till rapid emaciation, frequent "prune-juice" expectoration, absence of fever and of tubercle bacilli in the sputum, and, finally, the appearance of metastases, established the diagnosis of malignant disease. No autopsy was obtained.

The following cases were never considered to be tuberculosis, but are related because of the pulmonary signs and conditions.

Case 5.—*Malignant Disease of Lungs.*—Male, aged about 60; gradually increasing weakness, dyspnœa and dry cough; aspiration of blood-stained fluid from left chest; gradual

loss of weight; bloody sputum without tubercle bacilli; several aspirations. No autopsy.

Case 6.—Small Round-celled Sarcoma of First Rib, involving Lung and Pleura.—Widow, aged 68. Past history not important, except rheumatism for ten years and shingles nine years ago. Pain in left shoulder, accompanied by an eruption said to have been herpes; later, numbness and severe pain in left arm; lost 15 or 20 lb.; in bed most of the time for four months; slight dry cough.

Physical Examination.—Poorly nourished, sallow, dry and foul tongue. Temperature slightly elevated; evidence of arterio-sclerosis, with hypertrophied heart; slight dulness above and below left clavicle with diminished breath sounds; no râles. Liver somewhat large and tender. Marked tenderness in left supra-clavicular fossa, along inner side of upper arm, and front of forearm; some muscular atrophy and reaction of degeneration. Œdema of legs.

In two months it was noted that she was weaker; had greater paralysis of left arm and hand; that cough was increased, with slight expectoration, which was free from blood and tubercle bacilli, but contained elastic fibres; that dulness at left apex extended to second rib in front and to middle of scapula behind, with diminished breath and voice sounds. There were a few fine moist râles. Above clavicle were to be felt several small hard glands. Patient died eight months from first symptoms.

Autopsy showed a small round-celled sarcoma of the first rib, involving the pleura and apex of left lung, and pressing on the brachial plexus and vessels. It was densely adherent to all neighbouring structures. The first rib was largely disintegrated, from the spine to within $1\frac{1}{2}$ in. of the sternum. The right lung and pleura showed no change.

It seems, perhaps, worth while to report these cases, and particularly to discuss some of their symptoms and signs with reference to the differential diagnosis from pulmonary tuberculosis.

It is of value to compare the actual findings in cases, as they present themselves for observation to the practising

physician, with the more general accounts to be found in the literature.

It will be seen that they present problems in diagnosis which are by no means easy.

Most intrathoracic tumours are malignant, and what follows has chiefly to do with malignant cases.

Dr. Isaac Adler, in an excellent and recent monograph entitled "Primary Malignant Growths of the Lungs and Bronchi," quotes Williams as follows: "55 per cent. of all cases were carcinoma, 9 per cent. sarcoma, 25 per cent. non-malignant, and 11 per cent. cysts."

Our late and lamented Curtin, in his paper last year, quoted statistics indicating that primary cancer of the lungs occurs about once in 2,000 routine autopsies, or once in about 800 cases of cancer.

Babcock, in quoting several sets of autopsies, found one case in about 1,500 autopsies.

They may occur at any age, carcinoma being most frequent between the ages of 50 and 60; sarcoma between 30 and 40.

They are considerably more frequent in males. Secondary growths are very much more frequent than primary, and generally cause less symptoms relative to the chest, because they do not attain such size, are generally multiple, and their local manifestations are obscured by those of the primary disease and other metastases.

Sarcoma appears to be more frequent in the mediastinum, while carcinoma is very much more common in the bronchi and lungs.

It is probable that most cases of lung carcinoma originate in the bronchi, and that the pleura is involved as part of the process.

Whether the disease begins in the lungs or in the mediastinum, there is generally an extension from one to

the other during the course of the disease, so that the symptomatology is likely to be much the same in either case, except as to sequence.

All writers agree that there may be, in different cases, great variations in the relative prominence of different symptoms. There have been instances reported in which there have been hardly any symptoms till a sudden fatality.

The early symptoms in most cases are cough, expectoration, and dyspnœa.

Cough may be dry or with moderate expectoration. If there is obstruction of the trachea or large bronchi or pressure on the recurrent laryngeal nerves it is likely to be husky, rasping, or "brassy."

In general it is not troublesome and may be absent. It was a symptom in all of this series of cases.

Adler says if no cause can be found for a persistent cough, lung tumour should be thought of.

Expectoration may be absent or insignificant, but Adler considers it one of the most important things in diagnosis. In 10 per cent. of his 374 carcinoma cases there was no sputum; and in 40 per cent. it was not mentioned, being present in 50 per cent. It was present, though slight, in 83 per cent. of my cases.

Homogeneous, tenacious sputum of a colour resembling "currant jelly" is said to be characteristic; and Curtin laid stress on the appearance of globules of such sputum enclosed in a transparent gelatinous coating.

Occasionally there may be small masses of the tumour or cells in layers held together by a stroma, which may establish the diagnosis (Babcock). Lenhartz finds large spherical cells filled with multitudes of fatty granules and associated with many epithelial cells, which are curiously deformed and have club or tail-like projections.

He thinks these are characteristic of carcinoma, and that co-existent tuberculosis does not modify this feature.

Adler says that since his attention has been called to these cells he has found them in every case of primary carcinoma of the lung that he has seen, and he regards them as probably pathognomonic for carcinoma. He says, however, that their appearance is very inconstant, and that the sputum should be examined repeatedly.

Dyspnœa is often the first, and may be the most prominent symptom; yet, like all other symptoms, it may be absent. In Adler's cases of carcinoma it was present in 44 per cent., and not mentioned in 50 per cent. In ninety cases of sarcoma it was present in 60 per cent., absent in 2 per cent., and not mentioned in 40 per cent. It was present in 83 per cent. of my cases. It is apt to be progressive, and may be most distressing. When severe it is often paroxysmal. Death may occur from suffocation, due to compression of the larger air passages. The worst dyspnœa occurs with mediastinal growths, and is therefore more common in sarcoma, which is more frequent here.

Pressure symptoms may be of much aid in diagnosis. Obstruction to venous return causes dilatation of the veins of the head, neck, chest, or arms, and there may be œdema. There may be hoarseness or loss of voice from pressure on the recurrent laryngeal nerves, inequality of the pupils, or there may be dysphagia from pressure on the œsophagus.

Babcock and J. N. Hall mention the occurrence of "tracheal tug" and pulsation in cases of tumour adherent to the trachea, or located under the aortic arch and above the bifurcation of the trachea.

There were pressure symptoms in 50 per cent. of these cases.

In *Case* 3 the pulsation of the shadow seen by the fluoroscope led to the suspicion of aneurism.

Pain is often absent, and generally only a discomfort. The occurrence of much pain means the involvement of the

pleura or pressure on nerve trunks, for the lung parenchyma is not sensitive to pain. It may be referred to different localities.

Babcock says that marked persistent pain in the chest, without evidence of pleurisy, is probably not due to tuberculosis, and may be from tumour.

Pain, in a slight degree, was present in only 33 per cent. of this series.

Pleural involvement and effusion generally occur in malignant cases. The fluid may be clear, sometimes fatty from disintegrated tissue, but is likely to become bloody, as shown by repeated aspirations. This also occurs in tuberculosis.

The amount of fluid is generally small, is sacculated, and tapping rarely gives much relief from dyspnœa and other symptoms.

After aspiration there may be opportunity to make out conditions that were obscured by the effusion; and Adler points out that the failure of a dislocated heart to show any tendency to return to its normal position after aspiration should lead to suspicion of tumour. Of course, fibrotic and pleural contractions on the opposite side of the chest might lead to permanent dislocation of the heart.

There was pleural effusion in half of my cases.

Fever is not constant, and is irregular in character. This may be of some help in distinguishing it from tuberculosis. In all of my cases there was more or less fever, irregular in type. It is probable that the fever is chiefly caused by complicating conditions.

Night Sweats are said to be uncommon, occurring in 33 per cent. of this series.

The Blood Count appears not to have been well worked out, but there has been a fairly constant leucocytosis in the reported cases when the blood count was mentioned. It is

possible that here may be some help in differentiating tuberculosis. There was a slight leucocytosis in one-third of my cases. In two-thirds no record of this detail was made.

Course and Termination.—The disease may run its course in from a few weeks to five or six years. It is always fatal, death occurring from asthenia or suffocation.

The Physical Signs, of course, vary greatly according to the location and extent of the disease, the pressure on different structures, and the existence of complications. Early in its course there may be no signs at all. If the mediastinum and roots of the lungs are much affected, there may be some of the pressure effects already mentioned. In *Cases* 1 and 3 there was enlarged liver and spleen from passive congestion. *Case* 1 also showed dilated veins.

There is likely to be dulness over the sternum or interscapular spaces, and there may be a prominence, perhaps with pulsation of tumour, either palpable or visible by the fluoroscope, as in *Case* 3. If the growth obstructs a bronchus, there may be signs of the atelectasis and bronchiectasis in the lung and bronchi beyond. These signs may occur also if the disease is confined to the lung and bronchi.

The heart may be displaced, and there may be pleural effusion, which will complicate all findings. Enlarged glands in the neck, axilla, or other metastases are likely to occur in late cases.

Respiration over the growth may be loud and bronchial in character, with exaggerated voice sounds, if the underlying or supplying bronchus is patent; or there may be absence of both if it is obstructed. The heart sounds are likely to be well conducted in either case.

The coincidence of poor transmission of voice and respiratory sounds, with good conduction of heart sounds, is a very suggestive sign.

Babcock speaks of Fraenkel's observation of the transition

from dulness to tympany and the return to dulness over the same area.

There may be signs of cavity, due either to excavation or to bronchiectasis. There may be all the physical signs of pulmonary tuberculosis. The lesion is more likely to be centrally located than in tuberculosis, and the X-ray shows its outline to be more definite.

Direct examination of the bronchi through the bronchoscope and the excision of pieces of the tumour have established the diagnosis in some cases.

Coincidence of Tuberculosis and Malignant Disease.—Rokitansky believed that tuberculosis and carcinoma were incompatible diseases, but it has been shown that the two conditions are more or less frequently associated in the lungs as elsewhere in the body. Adler found this to occur nineteen times in his series of 464 cases of carcinoma and sarcoma.

Weller, in the *Archives of Internal Medicine* for March, 1913, tabulates ninety cases of primary carcinoma of the bronchi, all submitted to autopsy. In eleven of these there was coincident pulmonary tuberculosis.

Kurt Wolf points out that bronchial carcinoma begins most frequently near the bifurcation of the larger bronchi, where there is most irritation from aspiration of dust, smoke, and other irritants; and that there are frequently found in these localities small pigmented lymph glands which are apt to be tubercular.

He advances the theory that these glands break down, perforate the wall of the bronchus, and may be sufficient local irritant to start carcinoma in persons predisposed to the disease. This theory, according to Adler, has not been generally accepted. It has also been claimed that the irritation of any chronic process, tubercular or not, may determine the incidence of cancer.

Differential Diagnosis from Tuberculosis.—We have seen that the two conditions may co-exist, and the diagnosis has been made during life in a few reported cases; but such coincidence is generally not recognizable.

In the early stages differentiation, as has been pointed out, may be extremely difficult or impossible; but in many cases some of the following points may lead to a correct diagnosis.

Age does not seem to be of much help, for we see both conditions at all ages.

The knowledge of reasons for lowered vitality, the occurrence of fever, night sweats, marked general symptoms and lesions at the apices or bases, with distinct moist sounds or evidences of excavation, point to tuberculosis.

On the other hand, early loss in weight, dry hacking cough, marked dyspnœa, persistent pain, the occurrence of pressure symptoms, dulness over the upper part of the sternum or upper interscapular spaces, with definite outline, as shown by X-rays; and particularly dulness, diminution or absence of respiratory murmur and voice sounds over an area where the heart sounds are well conducted, should lead to very strong suspicion of tumour.

Of course, finding tubercle bacilli in the sputum stamps the disease as tuberculosis; while the occurrence of the globules of bloody sputum, referred to by Curtin, and the cells described by Lenhartz, may go a long way toward establishing the diagnosis of tumour.

The repeated absence of tubercle bacilli and presence of elastic fibres in the sputum is suggestive, and too much importance cannot be given to careful and repeated examination of the sputum.

Unexplained leucocytosis should probably suggest tumour in an early case.

Bloody pleural effusion is more likely to indicate tumour,

but is not very infrequent in tuberculosis. Finding relatively small amounts of fluid, getting little relief from dyspnœa by aspiration, and failure of a displaced heart to tend to return to its normal position after tapping, are points strongly suggestive of tumour.

Small pieces of a growth brought out on the needle-point or found in the aspirated fluid may establish the diagnosis.

Displacement of the heart, apparently not caused by pleural effusion or cicatricial contraction, enlarged cervical or axillary glands or other metastases, give pretty definite evidence.

In conclusion it may be said :—

First.—Intrathoracic tumours are probably more frequent than has been supposed.

Second.—They are occasionally coexistent with pulmonary tuberculosis.

Third.—Early in their course they are likely to suggest tuberculosis, and differentiation may be impossible.

Fourth.—In cases of doubt it is very important that all hygienic measures for the treatment of tuberculosis be instituted at once pending a definite diagnosis.

THE INFLUENCE OF THE INJECTION OF TUBERCULIN ON THE EOSINOPHILE CELLS IN THE PERIPHERAL BLOOD.

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In 1904 I published the results of a study of the blood of twenty-five cases of pulmonary tuberculosis [1]. In the course of this study I noticed that in the fatal cases the eosinophile cells, as a rule, disappeared from the peripheral blood. In that paper I made the following statement: "I am inclined to believe that the presence or absence of eosinophiles in the peripheral blood of tuberculous patients is a sign of some prognostic significance." "The variation of the eosinophile cells in the circulating blood of tuberculous patients may be explained as follows: In cases of incipient tuberculosis, eosinophile cells are present in the blood-stream in about normal proportion, because there is not enough tuberculin manufactured in the lesions to produce a marked eosinophilia. When, however, the secondary infection with the organisms of suppuration occurs, the neutrophile cells are attracted by the chemotactic power of the products of their growth which circulate in the blood and the eosinophile cells are repelled, so that we find an excess of polymorphonuclear neutrophile cells in the peri-

pheral blood. As the patient gradually becomes more saturated with the latter poisons and the fatal termination approaches, the eosinophiles progressively diminish in number and disappear from the peripheral blood. If, however, the progress of the case is influenced favourably by treatment the symptoms of the secondary infection disappear and the chemotactic influence of the products of the pyogenic organisms is withdrawn, so that the tuberculin being produced in the lesions may exercise its chemotactic influence to attract the eosinophilous polymorphonuclear cells from the bone marrow into the blood-stream."

In 1907, in collaboration with Dr. Howard T. Karsner [2], I reported the result of a further study of thirty-one cases in which differential leucocyte counts were made in order to throw some light upon the prognostic significance of the eosinophile cells. As a result of this study we concluded that the eosinophile cells tend to disappear from the circulating blood as the progress of the disease brings the fatal termination nearer. We also concluded that if the patient improves under treatment, or if the disease shows a tendency to become arrested, the eosinophile cells reappear in the circulating blood.

If the hypothesis advanced in my first paper is correct the injection of tuberculin for diagnostic purposes ought to be followed by an eosinophilia. This result has been observed by Levaditi [3], Zappert [4], Bischoff [5], and Botkin [6]. Allen Smith, Mullen, and Rivas [7] have produced eosinophilia in the leucocytes of citrated dog's blood by the addition of tuberculin and other toxic materials. Ullom and Craig [8] and Wright and King [9] have not been able to see any prognostic significance in the eosinophile cells in tuberculosis. The former authors say: "At the beginning of the investigation the eosinophiles seemed to increase with the patient's improvement; but further

study did not support this view." The latter authors say that the eosinophile cells remain normal. Solis-Cohen and Strickler [12] found the percentage of eosinophile cells unaffected by the stage, or by the extent of the disease, or by the condition of the patient. On the other hand, Miller and Reed [10] have found that with the progress of the disease the eosinophiles become less numerous until they almost entirely disappear from the blood in the terminal stages. Rayevsky [11] found the eosinophile cells always decreased. The variation of the percentage of eosinophiles corresponded, in his experience, to the anatomical and histopathological changes in the lungs. The more extensive the process the lower the eosinophile percentage. In the far-advanced cases, with some exceptions, he failed to find them in the circulating blood.

At the time of the publication of my first paper, in 1904, I determined to investigate the influence of tuberculin when injected, on the eosinophile cells, for diagnostic purposes.

Case 1.—The first case in which I had the opportunity to study this matter was that of a boy, aged 10, a patient of Dr. Charles H. Frazier, in the University Hospital, Philadelphia. The child had double hip-joint disease of four or five years' duration. The left leg was ankylosed in abduction and flexion, with contraction of the knee. The right leg was ankylosed in abduction, with slight contraction of the knee; there were suppurating sinuses on both sides. The temperature ranged from 98° F. in the morning to 100° F. in the afternoon. Blood smears were made from this patient twice a day by Dr. B. A. Thomas, who was then Dr. Frazier's resident. The first smears were made the day before the first injection of 1 mg. of tuberculin was given. The second injection of 3 mg. of tuberculin was given two days later, and the third injection of 5 mg. of tuberculin was made two days after that. After the injection of the 5 mg. of tuberculin the eosinophile cells rose to 7·8 per cent. on the first examination, 6·2 per cent. on the second examination, and 6·4 per cent. on the third examination.

The injection of tuberculin was accompanied by an increase of temperature to 102·8° F., the highest noted. At the beginning of the examination of this patient the eosinophiles formed 2·2 per cent. and 3·2 per cent. of the leucocytes in the peripheral blood.

DETAILS.

January 18, 1905.—Temperature 98·2° F. First specimen of blood prepared at 10.45 a.m.

Polymorphonuclear neutrophiles	76·0	per cent.
Lymphocytes	17·8	,,
Transitionals	4·0	,,
Eosinophiles	2·2	,,
	100·0	,,

The red cells showed slight anisocytosis and deficient hæmoglobin. The second specimen of blood was prepared at 8.0 p.m. Temperature, 100·2° F.

Polymorphonuclear neutrophiles	55·2	per cent.
Lymphocytes	34·4	,,
Transitionals	6·8	,,
Eosinophiles	3·2	,,
Basophiles	0·4	,,
	100·0	,,

January 20, 1905.—One milligramme of tuberculin injected at 9 a.m. The third specimen of blood was prepared at 10.30 a.m. Temperature, 100° F.

Polymorphonuclear neutrophiles	68·0	per cent.
Lymphocytes	27·6	,,
Transitionals	2·8	,,
Eosinophiles	1·4	,,
Basophiles	0·2	,,
	100·0	,,

The fourth specimen was prepared at 5.45 p.m. Temperature, 101·6° F., the highest since the injection of tuberculin.

Polymorphonuclear neutrophiles	56·4	per cent.
Lymphocytes	34·8	,,
Transitionals	5·2	,,
Eosinophiles	2·4	,,
Basophiles	1·2	,,
	100·0	,,

January 21, 1905.—The fifth specimen of blood was prepared at 11 a.m. Temperature, 99·8° F.

Polymorphonuclear neutrophiles	72·0	per cent.
Lymphocytes	15·0	,,
Transitionals	8·0	,,
Eosinophiles	3·8	,,
Basophiles	1·0	,,
Myelocytes	0·2	,,
	100·0	,,

January 22, 1905.—Three milligrammes of tuberculin were injected at 9 a.m. Temperature, 10 a.m., 102·2° F. The sixth specimen of blood was made at 11.45 a.m. Temperature, 101·6° F.

Polymorphonuclear neutrophiles	75·0	per cent.
Lymphocytes	15·4	,,
Transitionals	7·2	,,
Eosinophiles	2·2	,,
Myelocytes	0·2	,,
	100·0	,,

January 23, 1905.—The temperature reached 102·8° F. at midnight yesterday, the highest after the injection of 3 mg. of tuberculin. The seventh specimen of blood was prepared at 10.15 a.m. Temperature, 99·2° F.

Polymorphonuclear neutrophiles	67·0	per cent.
Lymphocytes	18·2	,,
Transitionals	12·0	,,
Eosinophiles	1·8	,,
Basophiles	0·6	,,
Myelocytes	0·4	,,
	100·0	,,

The specimen contained many degenerated forms of leucocytes.

January 24, 1905.—Five milligrammes of tuberculin injected at 9 a.m. The eighth specimen of blood was prepared at 10.15 a.m. Temperature, 99·4° F.

Polymorphonuclear neutrophiles	62·6	per cent.
Lymphocytes	22·2	,,
Transitionals	6·8	,,
Eosinophiles	7·8	,,
Basophiles	0·4	,,
Myelocytes	0·2	,,
	100·0	,,

The ninth specimen of blood was prepared at 6 p.m. Temperature, 102° F.; pulse, 124; respiration, 28.

Polymorphonuclear neutrophiles	...	56·0 per cent.
Lymphocytes	...	32·4 ,,
Transitionals	...	5·0 ,,
Eosinophiles	...	6·2 ,,
Basophiles	...	0·4 ,,
		100·0 ,,

Nine degenerated eosinophile cells were seen in counting 500 leucocytes.

January 25, 1905.—Yesterday, at 6 p.m. and 9 p.m., the temperature was 102° F., highest reached after the injection of 5 mg. of tuberculin. The tenth specimen of blood was prepared at 10.45 a.m. Temperature, 99° F.

Polymorphonuclear neutrophiles	...	72·8 per cent.
Lymphocytes	...	14·8 ,,
Transitionals	...	5·6 ,,
Eosinophiles	...	6·4 ,,
Basophiles	...	0·4 ,,
		100·0 ,,

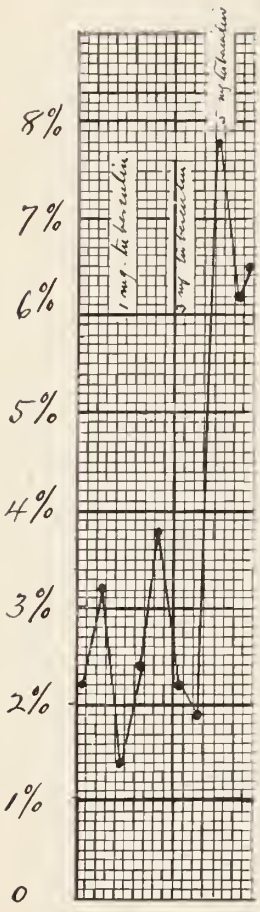
In this count only 250 leucocytes were counted.

Case 2.—The second case in which I had the opportunity to notice the influence of the injection of tuberculin on the eosinophile cells was that of a male, aged 65, who was supposed to have Addison's disease. The patient was being treated in the service of Dr. Alfred Stengel, for whom I was then substituting, in the Philadelphia General Hospital. This patient died shortly after the last dose of tuberculin was given, but before death tubercle bacilli were found in his sputum. No autopsy was obtained. In this patient the eosinophile cells disappeared from the blood after the first injection of 3 mg. was given, and did not reappear.

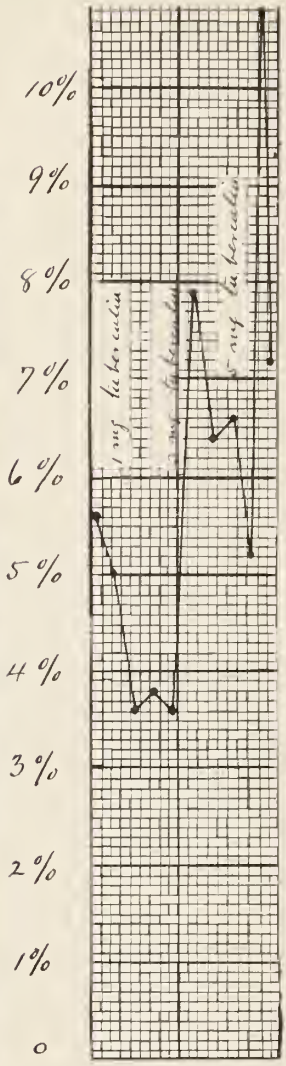
Details.

July 21, 1905.—The first specimen of blood was prepared at noon. Temperature, 97·8° F.; pulse, 105.

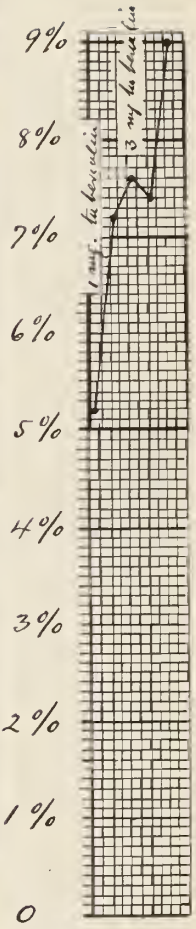
Polymorphonuclear neutrophiles	...	75·0 per cent.
Lymphocytes	...	14·4 ,,
Large mononuclears	...	7·2 ,,
Eosinophiles	...	0·4 ,,
Basophiles	...	1·2 ,,
Myelocytes	...	1·8 ,,
		100·0 ,,



CASE I.—Male, aged 10.



CASE III.—Female, aged 7.



CASE IV.—Male, aged 25.

The second specimen of blood was prepared at 4 p.m. Temperature, 97·8° F.; pulse, 105.

Polymorphonuclear neutrophiles	85·0	per cent.
Lymphocytes	9·2	,,
Large mononuclears	4·0	,,
Transitionals	0·2	,,
Eosinophiles	0·2	,,
Basophiles	0·4	,,
Myelocytes	1·0	,,
	100·0	,,

July 22, 1905.—Three milligrammes of tuberculin were injected at 10 a.m. The third specimen of blood was prepared at 11 a.m. Temperature, 98·4° F.; pulse, 104.

Polymorphonuclear neutrophiles	83·4	per cent.
Lymphocytes	11·2	,,
Large mononuclears	4·6	,,
Basophiles	0·8	,,
	100·0	,,

The fourth specimen of blood was prepared at 5 p.m. Temperature, 98·4° F.; pulse, 100.

Polymorphonuclear neutrophiles	78·6	per cent.
Lymphocytes	12·6	,,
Large mononuclears	8·2	,,
Basophiles	0·6	,,
	100·0	,,

July 23, 1905.—The fifth specimen of blood was prepared at 11 a.m. Temperature, 98·8° F.; pulse, 105.

Polymorphonuclear neutrophiles	80·2	per cent.
Lymphocytes	13·0	,,
Large mononuclears	6·4	,,
Transitionals	0·4	,,
	100·0	,,

The sixth specimen of blood was prepared at 5 p.m. Temperature, 99° F.; pulse, 110.

Polymorphonuclear neutrophiles	84·4	per cent.
Lymphocytes	9·8	,,
Large mononuclears	3·6	,,
Transitionals	0·6	,,
Myelocytes	1·6	,,
	100·0	,,

July 24, 1905.—Six milligrammes of tuberculin were injected at 10 a.m. The seventh specimen of blood was prepared at 10.45 a.m. Temperature, 98·4° F.; pulse, 120. The patient was perspiring freely.

Polymorphonuclear neutrophiles	88·4	per cent.
Lymphocytes	8·2	,,
Large mononuclears	3·2	,,
Basophiles	0·2	,,
	100·0	,,

The eighth specimen of blood was prepared at 5.20 p.m. Temperature, 97° F.; pulse, 100.

Polymorphonuclear neutrophiles	76·6	per cent.
Lymphocytes	9·2	,,
Large mononuclears	14·0	,,
Basophiles	0·2	,,
	100·0	,,

July 25, 1905.—The ninth specimen of blood was prepared at 12.30 p.m. Temperature, 96° F.; pulse, 122. The sputum contained tubercle bacilli.

Polymorphonuclear neutrophiles	88·6	per cent.
Lymphocytes	5·6	,,
Large mononuclears	5·2	,,
Basophiles	0·6	,,
	100·0	,,

The tenth specimen of blood was prepared at 6 p.m.

Polymorphonuclear neutrophiles	84·0	per cent.
Lymphocytes	7·6	,,
Large mononuclears	8·4	,,
	100·0	,,

Case 3.—The third case occurred in the person of a female, aged 7, who had hip-joint disease. She was a patient of Dr. James K. Young in the Polyclinic Hospital, Philadelphia (13532).

This case was interesting, because at the time of the first observation the patient had an eosinophilia due to the presence of *Ascaris lumbricoides* in the intestines. After the passage of the round worms the eosinophilia disappeared, and after the administration of the last dose of the tuberculin there was a marked increase in the number of eosinophile cells.

Details.

January 2, 1907.—The first specimen of blood was prepared at 3.30 p.m. Temperature, 98·2° F.; respiration, 26; pulse, 92.

Polymorphonuclear neutrophiles	51·0	per cent.
Lymphocytes	29·4	,,
Transitionals	8·8	,,
Eosinophiles	5·6	,,
Basophiles	0·2	,,
Myelocytes	5·0	,,
	100·0	,,

January 3, 1907.—The second specimen of blood was prepared at 1.30 p.m., and immediately afterwards 1 mg. of tuberculin was injected. Temperature, 99·6° F.; pulse, 96; respiration, 26.

Polymorphonuclear neutrophiles	55·6	per cent.
Lymphocytes	29·4	,,
Transitionals	6·0	,,
Eosinophiles	5·0	,,
Myelocytes	4·0	,,
	100·0	,,

January 4, 1907.—The third specimen of blood was prepared at 9 a.m. Temperature, 98° F.; pulse, 92; respiration, 24. The patient passed one *Ascaris lumbricoides* at 3.30 a.m.

Polymorphonuclear neutrophiles	50·2	per cent.
Lymphocytes	20·8	,,
Transitionals	21·0	,,
Eosinophiles	3·6	,,
Basophiles	1·0	,,
Myelocytes	3·4	,,
	100·0	,,

The fourth specimen of blood was prepared at 4.30 p.m. Temperature, 101·4° F.; pulse, 130; respiration, 28.

Polymorphonuclear neutrophiles	59·8	per cent.
Lymphocytes	24·6	,,
Transitionals	8·6	,,
Eosinophiles	3·8	,,
Basophiles	0·8	,,
Myelocytes	2·4	,,
	100·0	,,

January 5, 1907.—The fifth specimen of blood was prepared at 10 a.m. Temperature 98° F.; pulse, 96; respiration, 24.

The patient passed one *Ascaris lumbricoides* at noon. Three milligrammes of tuberculin were injected at 2 p.m.

Polymorphonuclear neutrophiles	29·2	per cent.
Lymphocytes	9·2	,,
Large mononuclears	55·2	,,
Eosinophiles	3·6	,,
Basophiles	1·6	,,
Myelocytes	1·2	,,
	100·0	,,

January 6, 1907.—The sixth specimen of blood was prepared at noon. Temperature, 98·6° F.; pulse, 112, respiration, 24.

Polymorphonuclear neutrophiles	48·4	per cent.
Lymphocytes	28·6	,,
Large mononuclears	13·6	,,
Transitionals	0·8	,,
Eosinophiles	7·8	,,
Basophiles	0·8	,,
	100·0	,,

The seventh specimen of blood was prepared at 6 p.m. Temperature, 100·4 F.; pulse, 100; respiration, 32. The patient passed one *Ascaris lumbricoides* at 4 p.m.

Polymorphonuclear neutrophiles	40·0	per cent.
Lymphocytes	32·0	,,
Large mononuclears	20·6	,,
Transitionals	0·6	,,
Eosinophiles	6·4	,,
Basophiles	0·4	,,
	100·0	,,

January 7, 1907.—The eighth specimen of blood was prepared at 1 p.m. Temperature, 98·8° F.; pulse, 100; respiration, 28. Five milligrammes of tuberculin were injected at 1.15 p.m.

Polymorphonuclear neutrophiles	43·8	per cent.
Lymphocytes	31·0	,,
Large mononuclears	15·8	,,
Transitionals	2·2	,,
Eosinophiles	6·6	,,
Basophiles	0·4	,,
Myelocytes	0·2	,,
	100·0	,,

January 8, 1907.—The ninth specimen of blood was prepared at 11.30 a.m. Temperature, 98·2° F.; pulse, 104; respiration, 28.

Polymorphonuclear neutrophiles	32·2	per cent.
Lymphocytes	41·4	,,
Large mononuclears	17·0	,,
Transitionals	2·8	,,
Eosinophiles	5·2	,,
Basophiles	1·0	,,
Myelocytes	0·4	,,
	100·0	,,

The tenth specimen of blood was prepared at 10 p.m. Temperature, 100° F.; pulse, 120; respiration, 26.

Polymorphonuclear neutrophiles	35·2	per cent.
Lymphocytes	41·6	,,
Large mononuclears	6·4	,,
Transitionals	3·8	,,
Eosinophiles	10·8	,,
Basophiles	1·6	,,
Myelocytes	0·6	,,
	100·0	,,

January 9, 1907.—The eleventh specimen of blood was prepared at 1.30 p.m. Temperature, 98·2° F.; pulse, 100; respiration, 24.

Polymorphonuclear neutrophiles	47·2	per cent.
Lymphocytes	31·8	,,
Large mononuclears	8·4	,,
Transitionals	4·4	,,
Eosinophiles	7·2	,,
Basophiles	1·0	,,
	100·0	,,

Case 4.—The fourth case occurred in the person of a white adult male, a patient of Dr. W. Campbell Posey, in the Wills Hospital, Philadelphia. The patient was suffering from tuberculous iritis. The eosinophiles increased from 5·2 per cent. to 9·0 per cent. after the injection of 3 mg. of tuberculin. After the injection of 5 mg. of tuberculin one specimen of blood was lost on account of poor technique, and the second specimen showed no eosinophile cells.

DETAILS.

December 26, 1907.—The first specimen of blood was

prepared at 5 p.m., and 1 mg. of tuberculin was injected immediately after.

Polymorphonuclear neutrophiles	65·8	per cent.
Lymphocytes	11·8	,,
Large mononuclears	9·0	,,
Transitionals	7·6	,,
Eosinophiles	5·2	,,
Basophiles	0·6	,,
	100·0	,,

December 27, 1907.—The second specimen of blood was prepared at 11 a.m. Temperature, 98° F.

Polymorphonuclear neutrophiles	68·4	per cent.
Lymphocytes	8·0	,,
Large mononuclears	14·0	,,
Transitionals	1·8	,,
Eosinophiles	7·2	,,
Basophiles	0·4	,,
Myelocytes	0·2	,,
	100·0	,,

December 28, 1907.—The third specimen of blood was prepared at 11 a.m. Temperature, 100·6° F. Three milligrammes of tuberculin were injected at noon.

Polymorphonuclear neutrophiles	61·6	per cent.
Lymphocytes	14·0	,,
Large mononuclears	11·0	,,
Transitionals	4·4	,,
Eosinophiles	7·6	,,
Basophiles	1·4	,,
	100·0	,,

In counting 500 leucocytes one normoblast was seen.

December 29, 1907.—The fourth specimen of blood was prepared at 5 p.m. Temperature, 99° F.

Polymorphonuclear neutrophiles	61·8	per cent.
Lymphocytes	14·8	,,
Large mononuclears	14·2	,,
Transitionals	1·8	,,
Eosinophiles	7·4	,,
	100·0	,,

December 30, 1907.—The fifth specimen of blood was prepared at 11 a.m. Temperature, 98° F.

Polymorphonuclear neutrophiles	...	50·8 per cent.
Lymphocytes	...	16·2 ,,
Large mononuclears	...	17·4 ,,
Transitionals	...	5·6 ,,
Eosinophiles	...	9·0 ,,
Basophiles	...	1·0 ,,
		100·0 ,,

December 31, 1907.—The sixth specimen of blood was prepared at 11 a.m., and at noon 5 mg. of tuberculin were injected. Temperature, 99° F.

This specimen of blood was spoiled through poor technique.

January 2, 1908.—The seventh specimen of blood was prepared at 11 a.m. Temperature, 101·2° F.

Polymorphonuclear neutrophiles	...	84·6 per cent.
Lymphocytes	...	13·2 ,,
Large mononuclears	...	2·0 ,,
Transitionals	...	0·2 ,,
		100·0 ,,

Twenty-four hours after the injection of the last dose of tuberculin the temperature reached 103° F. on three occasions.

Case 5.—The fifth case occurred in the person of a coloured adult female, who was a patient of Dr. W. Campbell Posey in the Wills Hospital, Philadelphia. The patient was suffering from tuberculous keratitis. The eosinophiles at the beginning of the observation formed 4·2 per cent. of the leucocytes in the circulating blood. There was an increase on one occasion to 8·4 per cent. after the first injection of 1 mg. of tuberculin. Twenty-four hours after the injection of 3 mg. of tuberculin the temperature began to rise and reached 103° F., its highest point, in a little over forty-eight hours.

December 26, 1907.—The first specimen of blood prepared at 5 p.m. One milligramme of tuberculin was injected immediately after.

Polymorphonuclear neutrophiles	...	35·4 per cent.
Lymphocytes	...	35·6 ,,
Large mononuclears	...	18·6 ,,
Transitionals	...	4·6 ,,
Eosinophiles	...	4·2 ,,
Basophiles	...	0·8 ,,
Myelocytes	..	0·8 ,,
		100·0 ,,

December 27, 1907.—The second specimen of blood was prepared at 11 a.m. Temperature, 98·8° F.

Polymorphonuclear neutrophiles	52·4	per cent.
Lymphocytes	26·4	,,
Large mononuclears	13·2	,,
Transitionals	2·6	,,
Eosinophiles	4·8	,,
Basophiles	0·6	,,
	100·0	,,

December 28, 1907.—The third specimen of blood was prepared at 11 a.m. Temperature, 98·8° F. Three milligrammes of tuberculin were injected at noon.

Polymorphonuclear neutrophiles	41·6	per cent.
Lymphocytes	24·2	,,
Large mononuclears	24·0	,,
Transitionals	1·2	,,
Eosinophiles	8·4	,,
Basophiles	0·6	,,
	100·0	,,

December 29, 1907.—The fourth specimen of blood was prepared at 5 p.m. Temperature, 101·8° F.

Polymorphonuclear neutrophiles	66·2	per cent.
Lymphocytes	18·4	,,
Large mononuclears	9·6	,,
Transitionals	2·6	,,
Eosinophiles	3·0	,,
Basophiles	0·2	,,
	100·0	,,

December 30, 1907.—The fifth specimen of blood was prepared at 11 a.m. Temperature, 102·2° F.

Polymorphonuclear neutrophiles	73·0	per cent
Lymphocytes	20·4	,,
Large mononuclears	3·2	,,
Transitionals	2·6	,,
Eosinophiles	0·8	,,
	100·0	,,

December 31, 1907.—The sixth specimen of blood was prepared at 11 a.m. Temperature, 100·0° F.

Polymorphonuclear neutrophiles	40·2	per cent.
Lymphocytes	12·8	,,
Large mononuclears	3·0	.
Eosinophiles	4·6	..
Degenerates	39·4	.
	100·0	..

This specimen of blood, on account of poor technique, showed a large percentage of degenerated and disintegrated forms.

January 2, 1908.—The seventh specimen of blood was prepared at 11 a.m. Temperature, 99° F.

Polymorphonuclear neutrophiles	...	40·8 per cent.
Lymphocytes	...	44·2 ,,
Large mononuclears	...	9·6 ,,
Transitionals	...	1·6 ,,
Eosinophiles	...	3·0 ,,
Basophiles	...	0·4 ,,
Myelocytes	...	0·4 ,,
		100·0 ,,

Case 6.—The sixth case occurred in the person of a white female, aged 44, who was a patient of Dr. James K. Young in the Polyclinic Hospital, Philadelphia (11972). The patient was supposed to have tuberculosis of the vertebral column. In this case there is no eosinophilia, and the tuberculin reaction was negative. There was no elevation of temperature after either injection of tuberculin. The highest temperature recorded after the 1 mg. dose was 99° F.; after the 3 mg. dose 99·2° F.; after the 5 mg. dose 98·8° F.

November 11, 1909.—The first specimen of blood was prepared at 9 a.m.

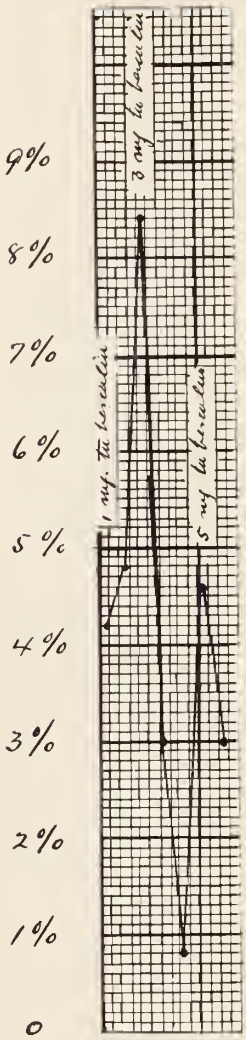
Polymorphonuclear neutrophiles	...	58·0 per cent.
Lymphocytes	...	31·4 ,,
Large mononuclears	...	3·6 ,,
Transitionals	...	3·2 ,,
Eosinophiles	...	3·8 ,,
		100·0 ,,

The second specimen of blood was prepared at 3.30 p.m.

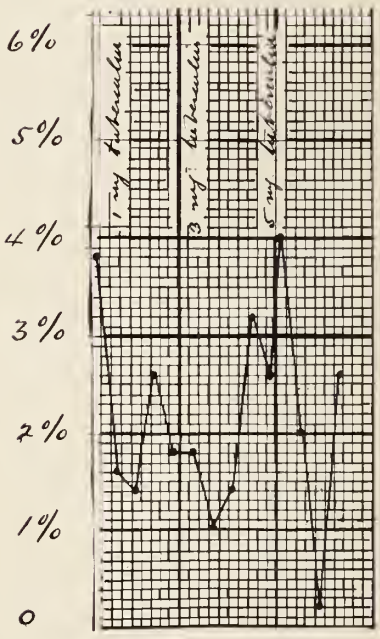
Polymorphonuclear neutrophiles	...	56·0 per cent.
Lymphocytes	...	38·8 ,,
Large mononuclears	...	1·0 ,,
Transitionals	...	2·2 ,,
Eosinophiles	...	1·6 ,,
Basophiles	...	0·4 ,,
		100·0 ,,

One milligramme of tuberculin was injected at 3.30 p.m.

November 12, 1909.—The third specimen of blood was prepared at 9 a.m.



CASE V.—Female, aged 25.



CASE VI.—Female, aged 44.

Polymorphonuclear neutrophiles	55·6	per cent.
Lymphocytes	36·6	,,
Large mononuclears	2·0	,,
Transitionals	3·8	,,
Eosinophiles	1·4	,,
Basophiles	0·6	,,
	100·0	,,

The fourth specimen of blood was prepared at 3.30 p.m.

Polymorphonuclear neutrophiles	57·6	per cent.
Lymphocytes	34·2	,,
Large mononuclears	2·6	,,
Transitionals	2·6	,,
Eosinophiles	2·6	,,
Basophiles	0·4	,,
	100·0	,,

November 13, 1909.—The fifth specimen of blood was prepared at 9 a.m.

Polymorphonuclear neutrophiles	58·8	per cent.
Lymphocytes	30·8	,,
Large mononuclears	5·0	,,
Transitionals	3·2	,,
Eosinophiles	1·8	,,
Basophiles	0·4	,,
	100·0	,,

The sixth specimen of blood was prepared at 4 p.m.

Polymorphonuclear neutrophiles	63·8	per cent.
Lymphocytes	28·0	,,
Large mononuclears	3·0	,,
Transitionals	3·4	,,
Eosinophiles	1·8	,,
	100·0	,,

Three milligrammes of tuberculin were injected at 4 p.m.

November 14, 1909.—The seventh specimen of blood was prepared at 9.30 a.m.

Polymorphonuclear neutrophiles	66·2	per cent.
Lymphocytes	24·8	,,
Large mononuclears	5·4	,,
Transitionals	2·6	,,
Eosinophiles	1·0	,,
	100·0	,,

The eighth specimen of blood was prepared at 3.30 p.m.

Polymorphonuclear neutrophiles	57·8	per cent.
Lymphocytes	37·2	,,
Large mononuclears	0·6	,,
Transitionals	3·2	,,
Eosinophiles	1·4	,,
	100·0	,,

November 15, 1909.—The ninth specimen of blood was prepared at 9 a.m.

Polymorphonuclear neutrophiles	62·0	per cent.
Lymphocytes	26·8	,,
Large mononuclears	2·4	,,
Transitionals	4·8	,,
Eosinophiles	3·2	,,
Basophiles	0·8	,,
	100·0	,,

The tenth specimen of blood was prepared at 6 p.m.

Polymorphonuclear neutrophiles	32·6	per cent.
Lymphocytes	60·0	,,
Large mononuclears	1·8	,,
Transitionals	2·6	,,
Eosinophiles	2·6	,,
Basophiles	0·4	,,
	100·0	,,

Five milligrammes of tuberculin were injected at 6 p.m.

November 16, 1909.—The eleventh specimen of blood was prepared at 9 a.m.

Polymorphonuclear neutrophiles	71·2	per cent.
Lymphocytes	18·8	,,
Large mononuclears	2·4	,,
Transitionals	3·4	,,
Eosinophiles	4·0	,,
Basophiles	0·2	,,
	100·0	,,

The twelfth specimen of blood was prepared at 4 p.m.

Polymorphonuclear neutrophiles	52·2	per cent.
Lymphocytes	39·0	,,
Large mononuclears	5·0	,,
Transitionals	1·6	,,
Eosinophiles	2·0	,,
Basophiles	0·2	,,
	100·0	,,

November 17, 1909.—The thirteenth specimen of blood was prepared at 9 a.m.

Polymorphonuclear neutrophiles	78·0	per cent.
Lymphocytes	15·8	,,
Large mononuclears	2·2	,,
Transitionals	3·0	,,
Eosinophiles	0·2	,,
Basophiles	0·2	,,
	100·0	,,

The fourteenth specimen of blood was prepared at 4 p.m.

Polymorphonuclear neutrophiles	51·4	per cent.
Lymphocytes	33·2	,,
Large mononuclears	8·0	,,
Transitionals	4·2	,,
Eosinophiles	2·6	,,
Basophiles	0·6	,,
	100·0	,,

Case 7.—The seventh case occurred in the person of a white male, who was a patient of Dr. A. A. Eshner and Dr. James K. Young in the Philadelphia Polyclinic Hospital (18215).

The first injection of 1 mg. of tuberculin was made at 3 p.m. on January 25, 1910. On January 26 the patient's temperature rose to 103° F., and the patient had an attack of tonsillitis. A culture made from the throat showed that the disease was due to infection with staphylococci and diplococci. No diphtheria bacilli were found. One milligramme of tuberculin was again administered on February 3, 1910, 3 mg. on February 4, 1910, and 5 mg. on February 5, 1910. There was no elevation of temperature after the administration of the increasing doses of tuberculin. The highest eosinophile percentage recorded was 4·0 forty-eight hours after the injection of 5 mg. of tuberculin.

Polymorphonuclear neutrophiles	55·0	per cent.
Lymphocytes	34·2	,,
Large mononuclears	1·8	,,
Transitionals	2·0	,,
Eosinophiles	6·4	,,
Basophiles	0·6	,,
	100·0	,,

One normoblast seen in counting 500 leucocytes.

January 25, 1910.—The second specimen of blood was prepared at 9 a.m.

Polymorphonuclear neutrophiles	84·4	per cent.
Lymphocytes	11·8	,,
Large mononuclears	0·2	,,
Transitionals	2·0	,,
Eosinophiles	1·2	,,
Basophiles	0·4	,,
	100·0	,,

The third specimen of blood was prepared at 3 p.m.

Polymorphonuclear neutrophiles	74·4	per cent.
Lymphocytes	16·4	,,
Large mononuclears	2·6	,,
Transitionals	4·4	,,
Eosinophiles	1·8	,,
Basophiles	0·4	,,
	100·0	,,

One milligramme of tuberculin was injected.

January 26, 1910.—The fourth specimen of blood was prepared at 9 a.m.

Polymorphonuclear neutrophiles	72·6	per cent.
Lymphocytes	18·6	,,
Large mononuclears	4·4	,,
Transitionals	3·0	,,
Eosinophiles	0·6	,,
Basophiles	0·8	,,
	100·0	,,

The fifth specimen of blood was prepared at 3 p.m.

Polymorphonuclear neutrophiles	75·0	per cent.
Lymphocytes	19·6	,,
Large mononuclears	1·2	,,
Transitionals	3·8	,,
Eosinophiles	0·4	,,
	100·0	,,

January 27, 1910.—The sixth specimen of blood was prepared at 9 a.m.

Polymorphonuclear neutrophiles	80·2	per cent.
Lymphocytes	14·0	,,
Large mononuclears	1·2	,,
Transitionals	3·6	,,
Eosinophiles	1·0	,,
	100·0	,,

February 3, 1910.—One milligramme of tuberculin was administered.

February 4, 1910.—The seventh specimen of blood was prepared at 9 a.m.

Polymorphonuclear neutrophiles	79·0	per cent.
Lymphocytes	13·8	,,
Large mononuclears	2·2	,,
Transitionals	3·0	,,
Eosinophiles	2·0	,,
	100·0	,,

The eighth specimen of blood was prepared at 3 p.m.

Polymorphonuclear neutrophiles	82·4	per cent.
Lymphocytes	14·8	,,
Large mononuclears	0·4	,,
Transitionals	1·6	,,
Eosinophiles	0·8	,,
	100·0	,,

Three milligrammes of tuberculin administered.

February 5, 1910.—The ninth specimen of blood was prepared at 9 a.m.

Polymorphonuclear neutrophiles	75·6	per cent.
Lymphocytes	19·2	,,
Large mononuclears	1·2	,,
Transitionals	2·2	,,
Eosinophiles	1·8	,,
	100·0	,,

The tenth specimen of blood was prepared at 3 p.m.

Polymorphonuclear neutrophiles	68·6	per cent.
Lymphocytes	21·0	,,
Large mononuclears	3·0	,,
Transitionals	5·4	,,
Eosinophiles	2·0	,,
	100·0	,,

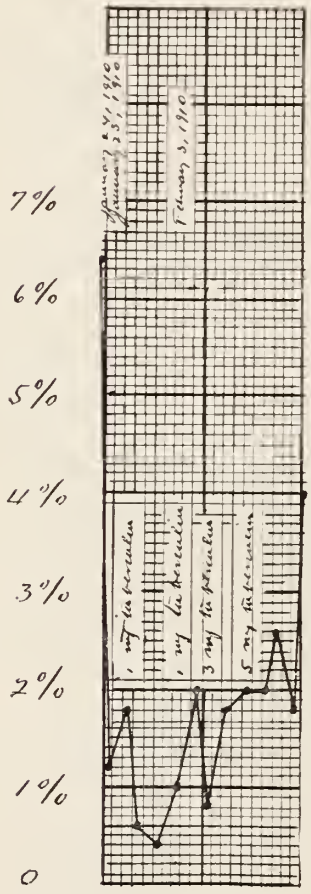
Five milligrammes of tuberculin administered.

February 6, 1910.—The eleventh specimen of blood was prepared at 9.30 a.m.

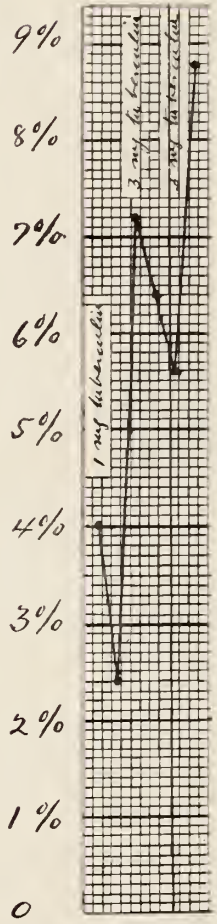
Polymorphonuclear neutrophiles	78·0	per cent.
Lymphocytes	12·4	,,
Large mononuclears	2·4	,,
Transitionals	5·2	,,
Eosinophiles	2·0	,,
	100·0	,,

The twelfth specimen of blood was prepared at 3 p.m.

Polymorphonuclear neutrophiles	74·4	per cent.
Lymphocytes	14·0	,,
Large mononuclears	5·4	,,
Transitionals	3·6	,,
Eosinophiles	2·6	,,
	100·0	,,



Case VII.—Male, age unknown.



Case VIII.—Male, aged 4.

February 7, 1910.—The thirteenth specimen of blood was prepared at 9 a.m.

Polymorphonuclear neutrophiles	82·4	per cent.
Lymphocytes	9·6	,,
Large mononuclears	2·4	,,
Transitionals	3·8	,,
Eosinophiles	1·8	,,
	100·0	,,

The fourteenth specimen of blood was prepared at 3 p.m.

Polymorphonuclear neutrophiles	75·6	per cent.
Lymphocytes	15·4	,,
Large mononuclears	1·0	,,
Transitionals	3·0	,,
Eosinophiles	4·0	,,
	100·0	,,

Case 8.—The eighth case occurred in the person of a male child, aged 4, who was a patient of Dr. Francis T. Stewart in the Polyclinic Hospital (15123).

The result of the tuberculin test was not typical. After the first injection of 1 mg. the temperature rose to 101° F. on January 26, 1908. On January 28, 1908, 3 mg. of tuberculin were given, but there was no elevation, the temperature being 99·4° F. On January 30, 1908, 3 mg. of tuberculin were again injected. This injection was followed by an elevation of temperature to 101·4° F. On January 31, 1908, 5 mg. of tuberculin were given, after which the highest temperature recorded was 99·2° F. In this case, after the injection of the second dose of 3 mg. of tuberculin, the eosinophile cells formed 8·8 per cent. of the leucocytes in the peripheral blood.

On January 26, 1908 :—

Polymorphonuclear neutrophiles	48·2	per cent
Lymphocytes	42·8	,,
Large mononuclears	3·0	,,
Transitionals	2·0	,,
Eosinophiles	4·0	,,
	100·0	

One milligramme of tuberculin was injected.

On January 27, 1908:—

Polymorphonuclear neutrophiles	62·2	per cent.
Lymphocytes	31·0	,,
Large mononuclears	2·2	,,
Transitionals	2·2	,,
Eosinophiles	2·4	,,
	100·0	,,

On January 28, 1908:—

Polymorphonuclear neutrophiles	44·2	per cent.
Lymphocytes	42·0	,,
Large mononuclears	4·6	,,
Transitionals	2·0	,,
Eosinophiles	7·2	,,
	100·0	,,

Three milligrammes of tuberculin were injected.
On January 29, 1908:—

Polymorphonuclear neutrophiles	44·0	per cent.
Lymphocytes	47·8	,,
Large mononuclears	1·2	,,
Transitionals	0·4	,,
Eosinophiles	6·4	,,
Myelocytes	0·2	,,
	100·0	..

On January 30, 1908:—

Polymorphonuclear neutrophiles	37·4	per cent.
Lymphocytes	51·8	,,
Large mononuclears	4·8	,,
Transitionals	0·8	,,
Eosinophiles	5·6	,,
	100·0	,,

Three milligrammes of tuberculin were injected.
On January 31, 1908:—

Polymorphonuclear neutrophiles	46·2	per cent.
Lymphocytes	35·4	,,
Large mononuclears	3·0	,,
Transitionals	5·8	,,
Eosinophiles	8·8	,,
Basophiles	0·4	,,
Myelocytes	0·4	,,
	100·0	,,

After this blood count was made 5 mg. of tuberculin were injected, but the differential counts made after this injection have been lost.

The tuberculin used in these cases was supplied to me by Dr. S. H. Gilliland, at that time Director of the Bacteriological Laboratories of the Pennsylvania State Live Stock Sanitary Board, except that used in Case 2, which was bought from a firm engaged in the manufacture of biological products.

My thanks are due to Dr. Charles H. Frazier, Dr. Alfred Stengel, Dr. James K. Young, Dr. W. Campbell Posey, Dr. A. A. Eshner, and Dr. Francis T. Stewart for permission to use their clinical material, and to Dr. Benjamin A. Thomas for assistance in making the smears from Case 1.

Summary.

This paper gives the details of the differential leucocyte counts in eight cases in which tuberculin injections were made for diagnostic purposes, in order to determine, if possible, the influence of tuberculin on the eosinophile leucocytes.

Case 1.—Eosinophiles before the injection of tuberculin, 2·3 per cent., 3·2 per cent.; after the injection of 5 mg. of tuberculin, 7·8 per cent. Tuberculin test, positive.

Case 2.—Eosinophiles before the injection of tuberculin, 0·4 per cent., 0·2 per cent.; after the injection of 3 mg. of tuberculin the eosinophiles disappeared from the peripheral blood, and remained absent until the patient died. Reaction, negative.

Case 3.—Eosinophiles before the injection of tuberculin, 5·6 per cent., 5·0 per cent. (*Ascaris lumbricoides* infection); after the injection of 5 mg. of tuberculin, 10·8 per cent. Reaction, positive.

Case 4.—Eosinophiles before the injection of tuberculin,

5·2 per cent.; after 3 mg. of tuberculin were injected, 9·0 per cent.; after 5 mg. of tuberculin were injected the eosinophiles disappeared. Reaction, positive.

Case 5.—Eosinophiles before the injection of tuberculin, 4·2 per cent.; after the injection of 1 mg. of tuberculin, 8·4 per cent. Reaction, positive.

Case 6.—Eosinophiles before the injection of tuberculin, 3·8 per cent., 1·6 per cent.; after the injection of tuberculin, no increase in the eosinophiles. Reaction, negative.

Case 7.—Eosinophiles before the injection of tuberculin, 6·4 per cent. and 1·2 per cent.; after the injection of 5 mg. of tuberculin, 4·0 per cent. Reaction, negative.

Case 8.—Eosinophiles before the injection of tuberculin, 4·0 per cent.; after the injection of 3 mg. of tuberculin, 8·8 per cent. Reaction, positive.

In these eight cases eosinophilia was seen after the injection of tuberculin for diagnostic purposes in five, and was absent in three. In the five cases in which eosinophilia was observed there was a positive reaction. In the three cases in which eosinophilia was not found the reaction was negative. One might draw two conclusions from these results: First, that eosinophilia is due to the fever accompanying a positive reaction. Second, after the injection of 5 mg. of tuberculin in a person who is not suffering from *Bacillus tuberculosis* infection, not enough tuberculin is administered to produce an eosinophilia.

The first conclusion must be rejected because all elevations of temperature are not accompanied by eosinophilia. The second conclusion may or may not be justified. I have no evidence to offer upon that point.

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